ARCHIVES OF OTOLOGY.

RE-EXAMINATION OF THE HEARING OF DEAF-MUTES ORIGINALLY TESTED IN 1893.

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With Plates I. and II. of Vol. XXX. of Germ. Ed.

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THE examinations of the hearing of deaf-mutes, which I made some years ago in Munich, have been carefully investigated by the Ministry of the Interior in the Kingdom of Bavaria, and the Minister of Education has lately given me an opportunity to demonstrate before him, personally, by means of the continuous series of tones as well as by the voice, the presence of some remnants of hearing in a number of scholars in our Deaf-Mute Institute. Whilst making this demonstration, I found that twenty-eight of the scholars whom I had tested before still remained in the Institute, and it then occurred to me to extend my former investigations into this very interesting province of otiatrics, by means of Prof. Edelmann's improved series of tones.

It was with a good deal of anxiety that I began this re-examination, for although I exercised the greatest care originally, the possibility of many errors having unintentionally crept in could not be absolutely excluded. For, in the first place, the tone-series originally employed was imperfect (especially in the twice-scored octave) in comparison with the other octaves, a defect which Prof. Edelmann has avoided by making much more intense the upper portion of the scale, the very space which is so often preserved in deaf-mutes. In the second place, I was afraid of the extreme youth, the defective replies, and lack of self-reliance in those

pupils who were now to face me for re-examination, for all the older pupils had graduated, only the youngest were left, and amongst the notes attached to their names, at the first examination, were such remarks as these: "Apparently total deafness," "Answers far from trustworthy," and so on.

However, in order to verify the condition of those who were then the youngest, to carry the question of the hearing of deaf-mutes still farther along, and for other reasons which shall soon appear, re-examination seemed not only desirable but even a necessity.

The first point to investigate was the different results likely to be obtained by Prof. Edelmann's new tone-series, with their powerful and reinforced tones in those regions especially in which my former series had been noticeably defective. Some authors, for example, have asserted that *equally powerful pure tones* do not produce the same results on the hearing when produced by different instruments. The falsity of this assertion I saw plainly enough at my original examination, and additionally I expressed the opinion that we might later on find some perception still present for a series of tones which had at that time escaped my observation, because in certain regions the intensity of my tone-series was too weak to get all possible results.

Furthermore, *we are not positively sure whether the remnant of hearing in deaf-mutes remains on the average entirely stationary.* We must assume in most cases *defects* in the percipient apparatus, between which a few districts are still preserved, and that the morbid process producing these defects has long since terminated. At the same time we can additionally represent to ourselves, that, for example, a cicatricial contraction, a calcification, or an ossification in the cochlea might, after years, extend in circumference and so destroy farther portions of the hearing spaces, particularly former islands of hearing. The inverse theory of return of the function by later *involution* must be regarded as extremely improbable, considering the basal morbid process, and the long time which has elapsed since it ceased to be active.

Finally, in answer to the question whether some defects

might not depend solely on torpidity of the auditory nerve elements, and might be improved by hearing exercises by means of tones corresponding with the defective regions, we can only reply that, judging from the successful results which Urbantschitsch and others think that they obtained by exercises with speech and simple tones, only practical experience can decide.

From such points of view the results obtained from the re-examination of these twenty-eight deaf-mute children seem important enough to guarantee a graphic representation, and comparison with the results originally obtained.

Much to my gratification, the variations are less than I thought they would be.

Plates I. and II., at the end of this number of the ARCHIVES, give a plain idea of the hearing remaining, the red lines showing what was originally present, and the blue, the results of the re-examination. The numerals with the ear named at the foot of both tables, as well as those in the remainder of this paper, refer to the cases successfully observed, and the same numbers refer to both examinations.

The greatest errors were discovered in the fifty-four ears belonging to twenty-seven re-examined children amongst those first noted as totally deaf.[1] For amongst these fifty-four ears twenty-five belonging to eighteen deaf-mutes were originally noted as totally deaf, whilst re-examination showed that four were by no means totally deaf, two having an island of hearing, and two an extensive district.

One island in Case 62 R (Plate I., Group I.) extended from a^{II} to a^{IV}, but was only discovered on blowing very forcibly with Edelmann's organ pipes, and the other one (38 L, Pl. I., Gr. I.) from f^{III} to e^{V}, and was only discovered by Edelmann's whistle and Lucae's forks c^{IV} to f^{IV}.

Case 73 R and L (Pl. II., Gr. IV. and VI.) was at first with difficulty tested and noted as "apparent totally deaf," and, although now exhibiting an extensive region for hearing, cannot be induced to speak. With exception of these four, twenty-one were totally deaf at both examinations.

[1] Case 28 was not amongst those first tested, and proved to belong to those having the best hearing.

On the contrary, amongst the twenty-nine ears which originally showed a remnant of hearing, only one was found in which formerly an island (c^{IV} to a^{IV}) was present, but which has at the re-examination given way to total deafness (Case 44 L, Pl. I., Gr. I.).

The right ear of this same patient (44 R, Pl. I., Gr. II.) is very interesting in comparison with the left, because in the former we found in the midst of a remnant of hearing, of about the same extent as originally with exception of a trifle at the lower tone-limit, *an island from* e^{II} *to* e^{III} *about as at first.* The patient's trustworthiness past and present, as demonstrated by the similar results obtained in the right ear, shows us that there can be hardly a doubt that the island of hearing originally present in the *left ear is now totally submerged.*

A somewhat similar case is seen in 39 L (Pl. I., Gr. I.), in which, at the first examination, two hearing regions were discovered, one from b^{III} to d^{V} and one from $f^{\sharp}$ to g^{I}, the latter disappearing in the interval between the two examinations. For this reason the case was taken from the group of defects and placed in that of islands. The upper end of the hearing region was *also abbreviated by seven semitones,* despite the more powerful forks employed, whilst in the other ear (39 R, Pl. I., Gr. I.) the island present at both examinations terminated at the second testing, just as originally at the upper end, whilst at the lower end, dependent on the more powerful sources of tone employed, it extended a little lower down.

These new defects probably signify an increased destructive process in Corti's organs, rather than an error in the examination.

Amongst twenty-eight ears there were but two in which the *original upper tone-limit exceeded the later by more than a semitone,* a difference which might be ascribed to mistakes in observation. In all the others, owing to the loudness of Edelmann's apparatus, the newly discovered regions for hearing *were a trifle more elongated than the old, not only at the upper tone-limit, but generally at all places where the hearing region is interrupted,* as well at the lower as the upper, or even at both ends.

I here emphasize the fact that *despite the great differences in power between the old and the new apparatus, just about the same tone-limits (or within a semitone) were found at both examinations.*

Leaving aside the examinations for Galton's whistle, which cannot be expressed in semitones, we find in twenty-eight ears that the region for hearing ceased at the same tone or within a semitone at the upper limit in seven cases, and at the lower limit in twelve cases, and, in the six cases with defects, twice at precisely the upper end of the defect, and twice at the lower. Moreover, there are eleven coincidences for Galton's whistle at the upper end of the hearing region. So that, taking all in all, thirty-four regions for hearing coincide substantially. Having found that the re-examination with the new apparatus exhibits the same tone-limits as those in the first examination, we must take it for granted *that a district of nerve elements provided with relatively normal functions joins directly on to another district which pathologically and anatomically has lost its former functions.*

These sharply defined pathological alterations are in all probability to be sought for in Corti's organs. They give us a well-defined picture of the situation of the districts destroyed, and we are justified in assuming that these pictures are better defined and more perfect than we could obtain by microscopic examination of the labyrinth, just as the determination of the visual field of the eye teaches us more precisely the form and extent of visual defects than a microscopic examination of the retina could do.

In contrast with this uniform coincidence of perception at thirty-four localities in twenty-eight ears, and partly at some other district in the same ear, we find more or less *increase* in the tone space discovered by the more intense new apparatus in twenty-six localities, the excess being but three times, more than one octave, averaging six tones, and from one to two *mm* by Galton's whistle.

Variations like these cannot be explained by mistakes in testing, but must be due to the greater power of the new apparatus. This condition must therefore appear at all

those spots where the transition from the hearing- to the deaf-region in Corti's organs is gradual, the result of diffuse destructive foci in the percipient organs.

Of the four regions which varied greatly three were in the same person (69 R and L, Pl. II., Gr. IV. and V.), and in the fourth (26 R, Pl. I., Gr. I.) Edelmann's fork and organ pipe were only heard when blown or struck most forcibly. Besides this, the lower portion of the newly discovered region lay in the weakest portion of my first tone-series.

The chief differences lie in the *lower border* of the region for hearing, the one in which *middle-ear processes*, tubal catarrh, etc., in children with hearing, good, bad, or none at all, chiefly exercise their morbid influence, and it may be that this disease was present in some cases at the original examination, or, for reasons cited in my former paper, these deaf-mutes were tested by aërial conduction alone.

The result of the re-examination with new instruments may be summed up in this way: *The number of totally deaf is less than before. Two deaf-mutes, however, lost considerable hearing in the interval, and it would seem as if we were justified in assuming that some cases always show slow advance of the destructive processes in the cochlea.* Two children had *more hearing* than at first test, which may be ascribed to defective replies originally in one case, and in the other to the greater power of Edelmann's apparatus. *The other twenty deaf-mutes showed about the same hearing at both tests, or a moderate increase averaging six semitones.*

The first and very frequent condition (similar, or nearly similar limit for tones) proves that the boundaries of morbid foci on the cochlea are often sharply defined. The second condition (a moderate increase in the extent of the region for hearing) gives us an approximate idea of the amount to which our results may be influenced by differences in the intensity of the various tone-series employed. Nevertheless, this influence has been much less than I had expected.

Although the re-examination discovered a few serious errors that had been made at the first testing, yet the number of coincidences was so great that there can be no doubt that even the youngest deaf-mutes in our Institute can be safely employed

for the collection of statistics of the hearing power of deaf-mutes.

Urbantschitsch has expressed the opinion that even if deaf-mutes are exercised exclusively by speech the hearing for musical instruments and speech alike is improved, even without the use of any of the former; but this opinion I cannot endorse.

Methodic exercises with *tones* were also tried in a very appropriate case for three months with an absolutely negative result.

The tests for the voice, vowels, consonants, and words were limited to cases in which the extensive hearing-region obtained in the tests for tones seemed to promise some minimum hearing for speech. These cases also had been suggested as suitable for such exercises and had already received some brief instruction by that method through the ear. More or less comprehension for speech was found in about one patient in every four — that is to say, in seven children with nine ears, two belonging to the second group (44 R and 58 L), one to the fifth group (26 L), and six to the sixth group (58 R, 66 R, 78 L, 43 R, 41 R, and 41 L).

At the original examination of these ears four were deaf for all vocal sounds except the consonants P, T, R, which were perceived by tactile sensation; at the re-examination, none were so deaf. Originally, three had hearing for a few vowels and consonants, at the re-examination only two. At the original examination two had hearing for words, at the re-examination seven.

In my first paper I laid down the upper and lower limits in the tone-scale which might be lost for hearing without totally destroying the perception for voice, namely the small space between b^{I} and g^{II}, which is indispensable for the understanding of words by the ear. Amongst the scholars re-examined there were but two who had lost perhaps from one to three semitones at the upper border of this little space (58 L and 44 R). The first one possessed the sixth, which I regard as necessary for hearing speech, but lowered a semitone in the scale. He could understand the word "Eight" and the vowels A and E, with one ear, and with the other

ear the same vowels and all the numerals except Five and Six.

The other one (44 R, Pl. I., Gr. II.), who had lost the *three* upper tones of the sixth for speech, and who was also totally deaf in the other ear though having some hearing at the original test, could repeat such numerals as Seven, Nine, Four, Twenty, and so on when spoken directly into the ear, *but could not understand any vowel except U.* Leaving aside the island from f^{II} to e^{III}, this child has a very extensive range of hearing, and an *unusually long duration for sounds.* From two points of view the case is interesting; for, firstly, we see how well speech was understood despite the loss for all vowels but U. We must assume that the numerals were recognized solely by combinations of consonants, despite the fact that all of the hissing sounds must have been more or less lost. The case is suggestive of what can be obtained in attentive children by instruction with the voice.

In the second place, we emphasize the fact that the vowel U was perceived although within the patient's defective regions lies the district d^{II} e^{II} in which Hermann has lately located[1] one of the fundamental tones for U, in opposition to Helmholtz, who located the only fundamental tone for U in the small f, which happens to lie inside the child's region for tones.

I will next refer to the hearing for the voice, in its relation to the situation of the regions for hearing in the tone-scale. For the *only case* which seemed incomprehensible at the first examination was 39 R (Pl. I., Gr. I.), in which both A and O were correctly repeated although the island for hearing lay far away from the fundamental tones for these vowels. The same island was found at the re-examination though elongated several semitones downward. Nevertheless A and O were no longer perceived, so that there must have been a mistake at the first test.

A single case (26 B, Pl. II., Gr. V.) with hearing from f^{II} to the middle of Galton's whistle, and which was at the first examination deaf for speech, now perceived the vowel I and

[1] *Arch. f. d. Gesammt. Physiolog.*, vol. liii., 1893.

the hissing sound "Sh," a condition which coincides with the position of the fundamental tones for the hissing sound, and with the upper fundamental tone, at least, of the vowel I.

The sixth group with extensive ranges for hearing is of great importance so far as future practice with the voice in partial deafness is concerned. Here we found six ears in five patients, one case, No. 73, being omitted, as it was not tested originally. Basing our opinion on the extensive regions for hearing, showing only slight defects at the upper and lower ends, it would seem as if such cases did not represent labyrinthine destruction in the least, but cerebral disturbances possibly in the auditory spheres in the temporal lobe, and that they therefore indicate "*word-deafness*," in the actual sense of the term. Experience alone can tell us whether such a central force of deafmutism is accessible to instruction through the ear. The slight results so far obtained are encouraging.

Four cases in Group VI. (58 R, 66 R, 78 L, and 43 R) were at first deaf to words, but re-examination shows an extensive comprehension for words, two repeating nearly all the numerals and two all of them correctly. The fifth child (41 R and L), who could originally perceive all the numerals, can now repeat them correctly across a room and even Latin words which are wholly unknown to him.

This slight experience proves that even the deaf-mutes of the VI. group, characterized by excessively defective comprehension for speech despite extensive perception for the tone-scale, are very accessible to instruction by speech through the ear. We know that wherever a satisfactory remnant of hearing exists, it can be utilized for the comprehension of the voice by well-conducted instruction, no matter whether the pathological alterations producing the defects of hearing lie in the cochlea or at any locality beyond.

One of the above-mentioned pupils (66) had on the other (left) side a very extensive region for hearing, with, however, a large defect in the middle, embracing the hearing Sixth (f^{I} to g^{II}), but if he closed with his moistened forefinger the right ear, belonging to the sixth group and hearing all the

numerals, he was unable on the side containing the defect to hear or to repeat a letter or word. The same thing was noticed in 43 and 78, who on the other side were not only deaf to tones but to speech.

This observation shows that closure of the meatus with the finger in deaf-mutes is sufficient to exclude all hearing from the other ear, even if it possesses abundant remnant of hearing. So that all tests of each separate ear may be considered as more reliable in deaf-mutes than in those who are partially deaf.

Case 78 shows that a part of the hearing for speech discovered at the re-examination is due to the preliminary instruction with the voice through the ear, for the child had only been educated with units, could for that reason only repeat numerals up to ten, and failed to comprehend the higher numerals, although she possessed a region for hearing from the upper limit for Galton's whistle to the middle of the great octave.

Small as are the statistics at my command, the results obtained for the hearing of speech indicate the surprising achievements in the comprehension of speech which brief instruction may produce.

SUPPLEMENTARY REMARKS.

A few days after handing in the present MSS. I received from the Minister of Public Instruction an edict of great importance for the future instruction of deaf-mutes in Bavaria, dependent on their remnant of hearing and speech.

In brief the edict says that all deaf-mutes newly entering the Institute are to be carefully tested for what remnant of hearing they may possess, as well as for any remaining capacity for the comprehension of speech; that those who still remain in the Institute may at any convenient time be re-examined in similar lines by competent aurists; and that those who hear a little or who can speak in the least, in addition to general instruction shall, in special hours, receive proper and skilled instruction for the preservation and possible increase of what hearing and speech they still possess.

Henceforward, then, we may surely expect that the special

care thus assured to the partly hearing and partly vocal deaf-mutes in Bavaria will soon be imitated in other parts of Germany, and that in a future not distant deaf-mutes of all countries will obtain instruction not only to increase what hearing and speech they may have, but this fraction of the normal hearing power to be utilized as a foundation for understanding spoken language such as now prevails, according to Mygind, in several institutions in America, France, and Austria.

FOURTH REPORT ON THE PATIENTS TREATED DURING THE YEAR 1898 IN THE HOSPITAL AND OUTDOOR DEPARTMENT FOR AURAL DISEASES AT THE UNIVERSITY OF STRASSBURG.

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Abridged Translation by Dr. Max Toeplitz, New York.

THIS report is based upon the same principles as that of the years 1896 and 1897. The abundance of the material rendered the writing of a record of the numerous small operations performed at the outdoor department impossible. The more important cases only will be given in this abstract. The character of the tumors, which are not classified, was not ascertained by exploratory excision and microscopical examination.

OUTDOOR DEPARTMENT, 1898.[1]

I.—AURAL DISEASES.

Disease.	Right.	Left.	Both.	Total.
A. External Ear.				
a. Auricle.				
Perichondritic abscesses	2	..	..	2
Eczema	22	14	11	47
Othæmatoma	..	1	..	1
Carcinoma	1	1	..	2
b. External Auditory Meatus.				
Exostoses	1	1	2	4
Cerumen	43	55	181	279
Foreign bodies	14	8	..	22
Otitis externa, circumscribed	27	52	1	80
Otitis externa, diffuse	18	21	8	47
Papilloma	1	1	..	2
Periauricular abscess following furuncle	..	1	..	1

[1] Unimportant diseases omitted in the translation.

Disease.	Right.	Left.	Both.	Total.
c. Membrana Tympani.				
Myringitis hemorrh. traumat.	1	..	..	1
Hemorrhages	3	4	..	7
Ruptures	3	9	..	12
B. Middle Ear.				
Hæmatotympanum	2	..	..	2
Otitis media, catarrhal acute	49	33	20	102
Otitis media, hemorrhagic acute	4	5	3	12
Otitis media, catarrhal chronic, and sclerosis	5	4	285	294
Otitis media, purulent acute	100	97	33	230
Otitis media, purulent acute with ostitis of mastoid process	8	8	..	16
Otitis media, purulent chronic	91	93	81	265
Otitis media, purulent chronic with polypi	22	12	5	39
Otitis media, purulent chronic with caries	4	13	1	18
Otitis media, purulent chronic with cholesteatoma	10	8	1	19
Sequelæ to Otitis media purulenta	10	6	28	44
C. Inner Ear and Nervous Disturbances.				
Otalgia	14	15	7	36
Neuralgia of the mastoid process	..	1	1	2
Injury to labyrinth	..	1	4	5
Other labyrinthine diseases	2	..	17	19
Fracture of base of skull	..	..	3	3

II.—DISEASES OF THE NOSE AND PHARYNX.

A. External Nose.

Disease	Total.
Eczema	38
Acne rosacea	2
Furuncle	9
Lupus	2
Erysipelas	1
Papilloma	1
Carcinoma	1

Disease.	Right.	Left.	Both.	Total.
B. Nasal Cavity.				
Rhinitis, catarrhal chronic	..	..	113	113
Rhinitis, hypertrophic chronic	4	..	76	80
Rhinitis, atrophic chronic	1	2	82	85
Ozæna	..	..	..	64
Lues nasi	..	..	11	11
Lues nasi congenital	..	..	4	4
Papilloma of the turbinated bodies	2	1	..	3
Polypi	6	12	19	37
Hypertrophy of the turbinated bodies:				
a, of the middle turbinated body	5	4	2	11
b, of the inferior turbinated body	1	2	1	4
c, of the posterior extremities	2	..	4	6
Empyema of Highmore's antrum	3	6	1	10
Empyema of all accessory cavities	3	1	1	5

Disease.	Right.	Left.	Both.	Total.
C. Naso-Pharynx and Pharynx.				
Rhino-pharyngitis, chronic	..	..	..	57
Rhino-pharyngitis, chronic atrophic	..	..	..	53
Pharyngitis, chronic	..	..	..	29
Pharyngitis, granular	..	..	..	26
Lues of pharynx	..	..	..	4
Lues of soft palate	..	..	..	1
Congenital luic defect in the soft palate	2	..	..	2
Uvula bifida	..	..	..	1
Paresis of velum palati	..	..	..	3
Papilloma of the soft palate	1	1	..	2
Cavernoma of the soft palate	..	..	1	1
Diphtheria of nose and pharynx	..	..	..	1
Hypertrophy of faucial tonsils	4	3	17	24
Hypertrophy of faucial and pharyngeal tonsils	..	..	..	183
Hypertrophy of the pharyngeal tonsil	..	..	..	480
Hypertrophy of the lingual tonsil	..	..	..	3

HOSPITAL OPERATIONS.

Name of Operation.	Number.
Incision of lymphadenitic abscesses	1
Incision of periauricular abscesses after furuncle	2
Extirpation of auricular tumors	3
Extirpation of aural polypi	1
Paracentesis	1
Chiselling of Mastoid according to *Schwartze*	15
Chiselling in perisinuous abscesses	9
Chiselling in extradural abscess of both cranial fossæ	1
Radical operation according to *Zaufal-Jansen*	32
Radical operation according to *Stacke*	1
Radical operation according to *Zaufal-Jansen* with opening of sinus	1
Radical operation in cerebellar abscess	1
Radical operation with chiselling of labyrinth	1
Excision of carcinoma of external nose	1
Extirpation of carcinoma of left nasal cavity	1
Extirpation of nasal polypi	12
Extirpation of hypertrophied turbinated bodies	10
Extirpation of posterior extremities and hypertrophied turbinated bodies	4
Incision of perichondritic abscesses of the nasal septum	3
Ablation of deviations of nasal septum	14
Opening of Highmore's antrum through canine fossa	1
Opening of Highmore's antrum through alveola	7
Chiselling of frontal sinus	1
Curettement of sphenoid cavity and ethmoid cells	1
Removal of tumors of naso-pharynx with cold snare	3
Extirpation of faucial tonsils	5
Extirpation of faucial and pharyngeal tonsils	113
Extirpation of pharyngeal tonsil	251
Removal of tumors of vocal cords	1
Removal of tumor of Santorini's cartilage (with cold snare by autoscopy)	1

I may here add a report on an epidemic of erysipelas, which appeared in the hospital at two different periods

during the summer and late fall. Through an outside physician suffering from erysipelas of the head, the infection had been transmitted into the operating room. All precautionary measures, immediately used (disinfection of the operating room and its surroundings, and also of the patients' rooms with formaline, etc.), could not prevent the infection with erysipelas of the following nine patients operated upon from April 1, 1898, until April 1, 1899, among sixty-nine cases:

1. Adele St., æt. twenty-one. Admitted April 27, 1898. Diagnosis: Otitis media, chronic, bilateral, purulent, with polypi and caries of the left temporal bone. April 29th: Radical operation, L. She was seized on May 10, 1898, with erysipelas, which soon assumed a bullous character and was followed by severe nephritis. On May 18, 1898: Exitus letalis.

2. Emilie E., æt. thirteen. Admitted March 3, 1898. Diagnosis: Otitis media, chronic, purulent, of R E, with periarticular abscess of the joint of the right jaw. April 11, 1898: Radical operation with opening of the abscess. June 18, 1898: Erysipelas. June 19, 1898: Transferred to the Children's Department of the hospital; thence, after implication of the non-operated side with erysipelas, transferred back on July 9th—after recovery from erysipelas. August 3, 1898: Additional attack of erysipelas on the right side, of mild character and of three days' duration. August 9, 1898: Discharged for out-of-door treatment. Beginning of September: Recovery.

3. Magdalene Sch., æt. nineteen. Admitted on June 23, 1898. Diagnosis: Otitis media, purulent, acute, of R E with mastoiditis. June 25, 1898: Chiselling according to Schwartze. In the beginning, the course of the wound was without reaction. July 12, 1898: On account of rise of temperature and infiltration of the jugular region, another operation was performed. The sinus (no thrombosis) was exposed and a diseased portion in the apex was removed. July 14, 1898: Erysipelas. She was transferred to the Department of Internal Diseases and thence discharged on August 9th for out-of-door treatment. End of September: Recovery.

4. Magdalene L., æt. forty-seven. Admitted on June 28, 1898. Diagnosis: Otitis media, purulent, subacute, of R E, with acute mastoiditis. July 2, 1898: Chiselling according to Schwartze.

July 18, 1898: Erysipelas. She was transferred to the Internal Department and thence discharged on August 19, 1898, for out-of-door treatment. Beginning of October: Recovery.

5. Marie B., æt. thirty-one. Admitted November 19, 1898. Diagnosis: Otitis media, purulent, chronic, of L E, with polypi. November 23, 1898: Radical operation. December 5th: Erysipelas. She was transferred to the Internal Department of the "Bürgerspital," and thence was discharged for out-of-door treatment, which is still carried on; however, the wound cavity is now (July, 1899) almost entirely epidermized.

6. Emil O., æt. twenty-nine. Admitted November 24, 1898. Diagnosis: Periauricular abscess after furuncle. November 26th: Opening of the abscess. December 10th: Erysipelas. He was transferred to the Internal Department of the "Bürgerspital," and thence discharged for out-of-door treatment. Beginning of February: Recovery.

7. Ignaz B., æt. thirty-three. Admitted October 31, 1898. Diagnosis: Otitis media, purulent, chronic, L E. November 3d: Radical operation. December 25th: Erysipelas. He was transferred to the Internal Department and on January 17, 1899, transferred back to the Aural Department; on March 1, 1899, he was discharged as almost entirely cured for treatment by his own physician.

8. Martha St., æt. sixteen. Admitted January 17, 1899. Diagnosis: Otitis media, purulent, chronic, R E. February 17, 1899: Radical operation. February 19, 1899: Erysipelas. The patient was isolated at once. March 23, 1899: discharged for treatment by her own physician. She had then a small granulating portion in the wound cavity, which was otherwise entirely epidermized.

9. Emma L., æt. thirteen. Admitted January 21, 1899. Diagnosis: Otitis media, purulent, chronic, bilateral, caries of the left petrous bone, abscess behind the L E. January 23, 1899: Radical operation L. After cessation of the incipient rise of temperature on January 31, 1899, suddenly 40.2° C. On the following day, typical erysipelas of the left aural region; on February 14, 1899, implication of the right side. On March 18, 1899: Discharged for out-of-door treatment; the wound cavity is now almost entirely epidermized.

It is superfluous to give a detailed description of the course of the erysipelas in each separate case, since it almost

always presented the same or at least a similar picture. The temperatures varied between 38.8° C. and 40.9° C. The diseased portions of the skin were always intensely swollen, highly reddened, their surface shining and mostly painful to the touch; in one case (No. 3), the sensibility upon pressure was increased to an enormous hyperæsthesia. The eruption, which started from the operated wound and, in all cases, had also implicated the scalp, was always defined by the well-known sharp, often serrated, boundary line from the surrounding parts. In three cases the hair fell out profusely, in two cases almost entirely. In two cases (Nos. 2 and 9), even the non-operated side was implicated. The urine contained albumen in three cases; albumen, cylinders, epithelia, etc., in two cases. Vesicles (erysipelas miliare) were present in two cases (Nos. 7 and 9); bullæ (erysipelas bullosum), in one case (No. 1). This case, which ended fatally, began and ran its course under the severest symptoms. The patient was extremely delirious and apathetic from the third day of sickness. Extreme apathy was also at times present in three cases (Nos. 3, 4, and 5). At any rate the intensity of the disease decidedly decreased during the course of the epidemic. For the treatment of the wound during the erysipelatous disease, dressings moistened with a two-per-cent. solution of carbolic acid were almost exclusively used.

Ever since the Aural Department, from the beginning of this year, has two rooms which are completely isolated from the other sick-rooms and received the cases of Nos. 8 and 9 as soon as the very first signs of erysipelas presented themselves, until to-day, no erysipelatous disease has appeared.

A CASE OF BEZOLD'S MASTOIDITIS SECONDARY TO FACIAL ERYSIPELAS; OPERATION; RECURRENCE OF THE ERYSIPELAS WITHIN TWENTY-FOUR HOURS; CURE.

By JOHN DUNN, M.D., Richmond, Va.

On January 13, 1900, I was asked to see Mr. A., aged fifty-nine. Previous history as follows: about December 10, 1899, facial erysipelas developed. It began at the bridge of the nose, spread across his face, involving finally the whole scalp, including the external ears. About the 20th of December Mr. A. experienced severe pain in the left side of his head; three or four days later his left ear began to discharge, without, however, being followed by any diminution of the pain in the aural region. This continued with great severity until I saw him on January 13th. At this time the patient's general appearance did not suggest grave intracranial complications. Appetite excellent. Pulse 90. Temp. 99½°. He was, however, suffering intensely with pain in the region of the left ear, which was discharging copiously a whitish-gray fluid. The left mastoid region was so swollen and œdematous, and so sensitive to pressure that little information as to the condition of the bone beneath could be obtained by palpation. On either side of the sterno-mastoid was a large swelling extending two inches below the mastoid tip. As the patient had just finished a rather hearty dinner when I first saw him, he was not operated upon until the following morning at 9 o'clock, at which hour his pulse was 84; temp. 98½°. The operation revealed the fact that practically the whole of the mastoid process had been destroyed. A small portion of its external surface was present, and attached to the fibrous portion of the sterno-mastoid about the tip were a few spicules of bone. The inner plate was also extensively destroyed, laying bare a large area of dura mater, whose surface was much roughened.

An incision was made through the skin, including the posterior one fourth of the sterno-mastoid, about two inches below the tip. Through this hole I inserted my middle finger, passing it beneath the sterno-mastoid muscle into the hole left by destruction of the mastoid process. The attic was not examined; the operation being brought to a close after removal of the remaining roughened pieces of bone about the process, and all the more quickly as there was considerable bulging of the brain membrane into the large hole in the inner-table. I could get no history of symptoms pointing to sinus thrombosis, so left the sinus unexamined. The usual dressings were applied. At eight o'clock the next morning I saw Mr. A., who had passed a fairly comfortable night. At this time his pulse was 90; temp. 98⅘ °. At ten o'clock I received a message from the nurse that the temperature had gone up to 102 °. I at once went to the hospital and removed the dressings to find that the auricle was immensely œdematous, being nearly half an inch thick and fiery red. Erysipelas had set in. Its blush could be seen to extend about three quarters of an inch over the skin anteriorly to the auricle; posteriorly it had reached the lip of the wound. The whole of reddened area, including the auricle and the external auditory canal, which was nearly impervious from the swelling, down to the drum membrane, was painted over three times with pure carbolic acid. The whole surface was then left covered for twenty-four hours with gauze saturated in pure alcohol; the external auditory canal being filled with alcohol every two hours. Quinine and tincture of iron were administered internally. By four o'clock next morning the temperature was down to 99 °, which point it did not reach again during the course of the convalescence. The discharge from the external canal ceased entirely within forty-eight hours. The dressings of the mastoid wound were changed twice daily for three weeks. The wound, which steadily grew smaller, to-day, March 30, 1900, closed up finally.

The case has been reported because mastoiditis, occurring in the course of erysipelas, is rare, and because of the rapidity with which the recurrent attack of erysipelas subsided under the prompt and thorough use of carbolic acid and alcohol.

NASAL EMPYEMA AS AN ETIOLOGICAL FACTOR IN THE ESTABLISHMENT AND CONTINUATION OF POST-NASAL CATARRH AND CATARRHAL INFLAMMATION OF THE MIDDLE EAR, WITH AN ESPECIAL CONSIDERATION OF THE ENLARGEMENT OF THE POSTERIOR END OF THE MIDDLE TURBINATE AS A PREDISPOSING CAUSE.

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THE widespread prevalence of catarrhal inflammation of the middle ear and the relentless course it pursues when once established, make the study of the disease of fascinating interest. That chronic catarrhal inflammation of the middle ear is caused, or at least unfavorably influenced, by catarrhal diseases of the nose and naso-pharynx is a fact which has been accepted by otologists for many years, and it is the relation, or perhaps identity, of the catarrhal inflammation of the ears to the general, or local catarrhal condition of the respiratory tract, which we wish to study in this paper. We will not consider in this connection those diseases of the nasal mucous membrane which are closely allied to diseases of the skin, of which the relation between eczema and asthma furnishes an example. This omission is made with a full appreciation of the relation of such diseased conditions of the respiratory mucous membrane to the chronic inflammatory process of the membrane lining the middle-ear cavity, but it has seemed preferable at this time to deal with the more common diseased conditions which are usually known as catarrhal—*i. e.*, accompanied by a discharge. Nor shall we consider such diseases

at suberculosis, syphilis, and new growths of the upper respiratory tract only in so far as they are a causative factor in the establishment or continuance of the catarrhal discharge. With this brief explanation of the scope of this paper, we will proceed to the consideration of the catarrhal disease condition of the upper respiratory tract and the chronic catarrhal inflammations of the middle ear which are caused by it.

Politzer states what is very probably the general belief among otologists when he says that "The diseases of the naso-pharynx, and of the nasal cavities, are not only frequently the forerunners of affections of the middle ear, but also exert an important influence upon the course and results of these affections. Their knowledge and treatment appears the more important to the ear specialist, as in the middle-ear disease, by the occurrence or continuation of a naso-pharyngeal affection, the ear disease is continued, and the return to the normal prevented." The recognition and removal of adenoid vegetations from the naso-pharynx has given, by the relief of catarrhal inflammation of the middle ear in children, abundant evidence that disease of the naso-pharynx is closely associated with middle-ear disease, and in this particular class of patients the naso-pharyngeal disease is without doubt the cause of the ear disease. If the other disease conditions of the naso-pharynx bear the same relation to middle-ear disease that adenoids do, then the study of the naso-pharynx and nasal cavities is of the utmost importance. It is important to inquire in what way a naso-pharyngeal or nasal disease affects the middle ear. The generally accepted opinion has been that the aural disease is caused by an obstruction to nasal breathing, and the negative pressure (Toynbee experiment) resulting from this. It is now known that adenoids produce aural disease in many cases in which they do not cause nasal obstruction, and that it is the location of the adenoid growth in or around the Eustachian tube, rather than the interference with nasal respiration, that causes the middle-ear disease. The location of an adenoid growth, as well as the changes which it undergoes from time to time, are very well seen in cases of cleft

aladte. In these cases there is evidently no nasal obstruction, but the obstruction of the Eustachian tube by the adenoid growth can be plainly seen. In other diseased conditions of the naso-pharynx associated with middle-ear disease, the same relation may exist between the two, though perhaps not always in the same way, that exists between middle-ear disease and adenoids. Adenoids may cause Eustachian obstruction in a purely mechanical way, while in other naso-pharyngeal diseases the mucous membrane of the Eustachian tube, and often that of the middle ear, is affected by an extension of the disease of the naso-pharynx. It is very rare for the other diseases of the naso-pharynx to cause the purely mechanical obstruction that adenoids do.

The question naturally arises, what are the causes of naso-pharyngeal disease and what makes it so intractable? The naso-pharynx is subject to any of the acute diseases of mucous membranes in general, but these attacks should, and in most cases do, run the course of acute inflammation of the mucous membrane in other situations. Acute naso-pharyngitis is a self-limiting disease, and there is nothing about the naso-pharynx that should cause an acute disease to pass over into a chronic one. The drainage is good, being in fact an inverted basin, and there are no pockets to retain the products of inflammation and so act as a source of repeated infections. The only pocket that was ever seriously claimed to exist in the naso-pharynx was Thornwaldt's bursa, and this is now known to be a depression in a neglected adenoid. Neglected adenoids may also cause bands of tissue, which evidently limit motion in the upper part of the pharynx, but these bands can hardly be said to form pockets which could retain infective material. Chronic naso-pharyngitis is not often, if it is ever, the result of an acute attack, although the acute exacerbations occurring in the course of the chronic disease are very deceptive and often pass as acute primary naso-pharyngitis. Acute disease of the naso-pharynx is probably always of bacterial origin. This opinion is held by Lennox Brown and many other competent observers. The different ways in which infection may reach the naso-pharynx are, first, as a part of the

general involvement of the whole of the upper respiratory tract, in coryza, influenza, and the beginning of pneumonia; secondly, by infection from chronic nasal disease; thirdly, by an extension upwards of an acute inflammation of the oro-pharynx, and lastly from such general systemic infections as tuberculosis, syphilis, general septicæmia, and the exanthemata. In those cases in which acute naso-pharyngitis occurs during the course of an acute coryza, the naso-pharynx may be involved at once or the inflammation may exist in the nose several days before it affects the naso-pharynx. During an attack of acute naso-pharyngitis from whatever cause, the lymph tissue of the pharyngeal vault is involved, but the inflammation should run the course of inflammation in simular tissue elsewhere and end in recovery. This is undoubtedly the result in a large majority of the cases, and it would be the result in all if the lymph tissue of the naso-pharynx were not subjected to repeated reinfection. If the naso-pharynx is examined with the post-nasal mirror in cases of post-nasal catarrh, the mucous membrane will be found red, swollen, and uneven. This condition may involve the whole post-nasal space in acute cases, while in the chronic cases, the redness and swelling may be limited to circumscribed patches varying in size from a few millimetres to several centimetres in diameter. The lymph glands are always involved, not alone in the vault of the pharynx, but those on the posterior and lateral walls of the oro-pharynx as well, and appear as solitary follicles (follicular pharyngitis), as bands or stripes (lateral pharyngeal hypertrophy), or as a slightly raised granular surface. There is very little secretion to be seen except in those cases where an atrophic rhinitis has extended to the naso-pharynx or in cases in which the post-nasal catarrh has existed for years and the acinous glands have become involved. In the latter class of cases the discharge is thin, glairy, and very tenacious, and it often gives a shiny appearance to the mucous membrane. The cases which have a large amount of secretion in the naso-pharynx are either cases of atrophic disease or neglected cases of sinus disease. In those cases in which the pharyngeal vault is free from secretion the patient can

by sniffing and clearing the throat obtain more or less secretion, thus showing the source of the discharge to be in the nasal cavities. Except in the cases noted above and in cases where either syphilitic or tuberculous ulcers exist in the post-nasal space, there is no evidence that any considerable amount of the discharge in cases of post-nasal catarrh originates in any part of the pharynx. On the other hand, the appearance of the naso-pharynx, the absence of secretion on inspection, and the method of clearing the throat shows that the discharge comes from the nose and that the lymph tissue is infected by this discharge. In other words, the inflammation of the mucous membrane of the naso-pharynx and the involvement of the lymph tissue is a secondary disease caused by an irritant discharge from the nasal cavities. That the discharge from the nose is irritant we have abundant evidence in the excoriations around the nostrils and extending to the lips in cases of acute rhinitis and the purulent catarrhs of children. If a condition similar to that which is found in the naso-pharynx existed in any other part of the body, no one at the present day would consider it a primary disease, but every surgeon would at once search for the source of the infection.

The effect of this condition of the naso-pharynx on the ears is very evident, the ears are especially liable to suffer, indeed they can hardly escape, if the lymph tissue in or around the Eustachian tubes is involved by repeated infection from an irritant nasal discharge. There is every reason to believe that the inflammation of the naso-pharynx may extend to the middle ear itself, either by continuity of tissue or by the forcible blowing of the irritant discharge through the Eustachian tube into the middle-ear cavity. The appearance of the membrane of the middle ear in cases of chronic catharrhal inflammation, as described by Politzer, resembles so closely the appearance of the mucous membrane of the naso-pharynx in cases of post-nasal catarrh as to suggest a common origin. The same solitary follicles and the same circumscribed patches of red, raised, granular mucous membrane exist in both and would seem to indicate that the disease was the same in one situation as in the other. For

reasons that are obvious, the results of the inflammation are much more serious in the middle ear than they are in the naso-pharynx. It is not the purpose of this paper to discuss the pathological changes that take place in chronic catarrhal inflammation of the middle ear but simply to show the relation of the two diseases and to find a rational explanation of chronic post-nasal catarrh.

If the assumption is correct, that to establish a chronic post-nasal discharge, with all its attendant symptoms, it is necessary to have a pocket which acts as a reservoir to retain the products of inflammatory action, and further if the normal naso-pharynx does not contain such pockets then it is necessary to examine the surrounding parts in search of such pockets. If we do this we find a large number (the nasal accessory sinuses) which open into the nasal cavities as a common drainage way. In the normal condition these sinuses communicate by free openings with the nose and whatever secretion there may be from them is carried, together with the secretion from the other parts of the nasal cavity, by the action of the cilia, to the naso-pharynx. It is only when the sinuses become the seat of chronic disease that they assume any importance as an etiological factor in the production of the chronic catarrhal condition. Disease of these sinuses may be so extensive and so severe as to menace the health or even the life of the patient, or they may be so slight that the only symptom is an intermittent discharge into the naso-pharynx. Whether we accept the statement of Hajek that "the whole chain of catarrhal symptoms of the nose, naso-pharynx, larynx, trachea, bronchi, and of the lungs themselves is dependant upon nasal empyema, we must, I think, admit that there is a close relation between the disease of the accessory sinuses and chronic catarrh of the upper part of the respiratory tract. I am fully convinced that there is no other explanation of chronic catarrhal naso-pharyngitis but this. I came to this conclusion from clinical experience, some time before I saw Hajek's statement, and further observation has only confirmed me in this belief. A daily discharge from a nasal empyema passing through the naso-pharynx is sufficient to

cause all the symptoms of post-nasal catarrh and I know no other condition that is.[1]

To establish a nasal empyema it is necessary to have certain predisposing causes which may exist either singly or combined in a given case; they are:

First. Congenital defective formation of the bony walls of the nasal cavities, such as a middle turbinate which is strongly rolled outward, so leaving a small space for drainage, small openings to the cavities, cavities badly placed for drainage, and an extremely narrow nasal cavity.

Secondly. Partial or complete closure of the normal openings of the cavities. This stenosis may be the result of disease or of acquired deformity, such as polypoid degeneration of the mucous membrane and new growths of all kinds, syphilitic, tuberculous, and nonspecific osteitis and periostitis, a bullous middle turbinate, spurs, deviated septum, and general or local vascular or fibrous hypertrophy of the nasal tissue. The last condition is very often the result of nasal empyema, and cannot therefore in many cases be considered as a cause.

Thirdly. Unhealthy surroundings which increase the liability of infection. Also the virulence of the infection and often the lowered resistance of the patient.

If we examine the predisposing causes more in detail, we find that deviated septi and spurs are causes of nasal empyema, post-nasal catarrh, and catarrhal deafness only when they are so located or are of such degree that they interfere with the drainage from the accessory sinuses. It follows therefore that operations for the relief of these conditions will have a beneficial effect upon the deafness only in those cases in which the more affected ear corresponds to the more occluded nasal cavity.

The chief predisposing cause of nasal empyema is the position, size, and diseased condition of the middle turbinate or of the tissues surrounding it. The middle turbinate varies in size from a mere ridge to a body several centimetres

[1] When this paper was nearly ready to go to press I found that this opinion of the origin of post-nasal catarrh is held by Grünwald and Moritz Schmidt. Grünwald's *Nasal Suppuration*, 2d ed. page 108.

in thickness. It usually extends only to the posterior third of the nasal cavity, but when diseased it may reach as far as the Eustachian tube.

We expect to find nasal empyema in all cases of polypoid disease in the region of the middle turbinate and the same holds true of new growths in this region. After all the cases in which there is evident disease of or around the middle turbinate have been eliminated, there still remains a vast number of cases which have nasal empyema. These cases are caused by a middle turbinate which approaches too close to the nasal wall, by cells which are badly placed for drainage, or by focal disease within the cell itself. The space between the middle turbinate and the nasal wall, through which the drainage from the antrum, the anterior ethmoid cells, and frontal sinus passes, may be much narrowed and still serve its purpose until the cells and the nasal mucous membrane have been subjected to repeated and prolonged attacks of inflammation, or the first severe attack may establish a nasal empyema. An acute inflammation may entirely close the natural openings of the cavities and thus cause the products of inflammation to become encysted or, as occurs in a large proportion of the cases, after the acute congestion has partially subsided, the inflammatory thickening only partially closes the openings and thus allows a discharge of mucous or muco-pus to escape into the nose and naso-pharynx, either constantly or at irregular intervals. In many cases there is never any discharge visible in the nose on examination, because of the middle turbinate being so closely rolled outward that it forms a sort of gutter which conducts the discharge to the naso-pharynx without it ever appearing in the nose. In this way the discharge from the antrum of Highmore or from the anterior ethmoid cells or frontal sinuses may reach the naso-pharynx and appear as a post-nasal catarrh.

In this connection I wish to call attention to a malformation of the middle turbinate which is almost constantly present in cases of post-nasal catarrh. So far as I know the relation of this particular malformation of the middle turbinate to post-nasal catarrh has not previously been

reported. The malformation consists of an enlargement and downward prolongation of the posterior end of the middle turbinate.[1] It is often large enough to nearly or quite fill the space between the septum and the nasal wall. It rarely reaches to the lower meatus, but is confined for the most part to the middle meatus. Owing to its situation far back in the nasal cavity, and also at times to the presence of a tortuous or narrow nasal chamber or, what is still more common, the presence of hypertrophy of the nasal mucous membrane, it may be easily overlooked, unless it be searched for after the tissues have been shrunken by the application of cocaine. The enlargement of the middle turbinate often contains a cell of some size which may be the source of some discharge, but the larger part probably comes from the posterior ethmoid cells or the sphenoid sinus, although, as previously stated, the drainage from any one of the sinuses may be concealed by the middle turbinate. The openings of the posterior ethmoid cells and sphenoid sinus are partially and probably at times wholly closed by this part of the middle turbinate, and it thus acts as a predisposing cause for the formation of an empyema in them. The formation of a gutter by the middle turbinate which has been alluded to before should not be forgotten in connection with this enlargement of the posterior end. This part of the nasal cavity is inflamed during each attack of acute rhinitis, from whatever cause, and often the inflammatory process is not apparent in any other part of the nasal cavity. This is especially noticeable in those patients that suffer from a succession of colds during the winter and spring months. If this part of the nose is carefully examined during the progress of an attack of this kind, the enlargement of the middle turbinate will be found

[1] In many of these cases the anterior part of the middle turbinate is not developed, so that which I have described as a malformation is the only portion which is visible by rhinoscopic examination, but even in those cases in which the enlarged posterior end comprises the whole of the middle turbinal, an essential malformation exists. It is well to bear in mind that in these cases the drainage from the antrum, the anterior ethmoid cells, and the frontal sinus probably still takes place through the space between the middle turbinate and the nasal wall and that it would therefore be much nearer to the naso-pharynx than in the normal nose.

swollen and dusky red in color, the rest of the nasal cavity perhaps being free from all appearance of inflammation. This swelling gives rise to a sense of stuffiness and to more or less headache. As the attack begins to subside it is often possible to see a mucous or muco-purulent discharge flowing down around this part of the middle turbinate and this visible discharge may persist for some time. It is very probable that the repeated colds from which these patients suffer are not fresh attacks, but rather exacerbations of an already existing nasal empyema. A patient that has a collection of fluid in one or more of the accessory sinuses generally suffers from what he considers to be a succession of colds in the head. During the warm months or in an atmosphere that is practically free from germs, the nasal empyema may give only slight annoyance, but during the colder weather, or in unhealthy surroundings, it will be the cause of a persistent catarrhal condition of the upper respiratory tract. The presence of this inflammatory condition in the cells renders the patient more susceptible to attacks of acute rhinitis from infection, but by far the greater annoyance comes from increased secretion, which resembles a cold in the head, and which may last from a few hours to several days. This increase of secretion may be the result of anything that causes congestion, such as indigestion, constipation, draughts of air, wetting the feet, dust- or smoke-laden atmosphere, menstruation, etc.

It is just this class of patients that have chronic catarrhal inflammation of the middle ear and in whom the deafness is made worse by each increase in the inflammatory condition of the nose or naso-pharynx. The enlargement of the posterior end of the middle turbinate, which I have described, is constantly present in these cases and I have been able to tell by the presence or absence of inflammation in this part of the nose, whether the deafness was worse or better. The increase of the deafness is coincident with the increase of the inflammation of this part of the nose and it may precede the increase of the post-nasal discharge, probably for the reason that the congestion from the increased inflammation extends to the Eustachian tube at once, while the same congestion

may lock up the discharge for several days. I am convinced that the malformation of the middle turbinate, which has been described, is the predisposing cause of the nasal empyema which produces a large percentage of the cases of post-nasal catarrh and catarrhal deafness. The reasons for this conclusion are briefly, that it practically always exists in cases of post-nasal catarrh, that an inflammation here precedes by a few days or is coincident with an increase of the inflammatory process in the naso-pharynx and middle ear, that it is often the only part of the nasal cavity which is inflamed in cases of acute rhinitis, that it is often possible to see the secretion flowing down around this part of the middle turbinate, and finally that it is possible to cure long-standing cases of post-nasal catarrh and relieve the ears from the danger of repeated attacks of inflammation, by the removal of enough of this malformation of this middle turbinate to give good drainage to the cells which may be diseased.

If the predisposing causes exist what is necessary to establish a nasal empyema? Unquestionably bacterial infection. According to Hajek every inflammation of the accessory sinuses, either acute or chronic, is due to this cause. We will briefly pass in review the different diseases during the course of which infection of the accessory sinuses may occur. Among these diseases acute coryza takes the first rank on account of the frequency of its occurrence. While it cannot, as yet, be demonstrated with certainty that the so-called cold in the head is due to bacterial infection, nevertheless its evident contagiousness, its clinical course, complications, and sequelæ leave no doubt but that such is its origin.

Next in frequency to acute coryza comes influenza. Weichselbaum was the first to demonstrate the influenza bacillus in accessory sinus disease. Later Lindenthal found the influenza bacillus almost constantly present in the accessory sinuses during the course of influenza and he found that it alone was sufficient to produce pus without the admixture of any other bacteria. Lindenthal was also led to believe from his investigations that in many of the cases where other bacteria were found with the influenza bacillus, that they were a secondary infection.

Next in frequency after influenza come croupous pneumonia, scarlet fever, diphtheria, measles, typhoid fever, facial erysipelas, cerebro-spinal meningitis, variola, etc. (Hajek).

Fränkel has called attention to the frequent occurrence of accessory sinus disease during the course of croupous pneumonia, and his researches, together with those of Weichselbaum, clearly establish the connection between the two diseases. Fränkel also found the pneumococcus of Friedlander in the normal accessory sinus.

The relation of diphtheria of the throat or nose to sinus disease presents itself in one of two ways—either there may be a true diphtheritic membrane formed in the accessory sinuses, particularly in the antrum (Weichselbaum, E. Fränkel, Dmochowsky), or the sinuses may be intensely inflamed during the course of the diphtheritic attack without true diphtheritic infection, the inflammation in these cases being due to a secondary infection by other bacteria (Zuckerkandl).

The relation between facial erysipelas and sinus disease has been observed and reported by Zuccarini, Weichselbaum, Zuckerkandl, Killian, Grünwald, Hajek, and others. Whether the facial erysipelas or the sinus disease is the primary disease has not as yet been settled. It is quite probable that one may be the primary disease in one patient and the other in another. The other bacteria most often found either alone or accompanying the influenza bacillus, in accessory sinus disease, are the staphylococcus pyogenes aureus and albus, the streptococcus pyogenes and the bacillus coli. (Weichselbaum, E. Fränkel, O. Lindenthal.) Whether the inflammation of the accessory sinuses occurring during the course of the above-mentioned diseases is due to the primary or to secondary infection by other bacteria, has not as yet been definitely settled only so far as relates to pneumonia, influenza, and diphtheria.

The frequent occurrence of nasal empyema as a sequel of scarlet fever and diphtheria is very noticeable. Equally so is the empyema following what is evidently a purulent rhinitis in children. Children may have a purulent rhinitis even from birth, as, perhaps, the nasal mucous membrane may be

infected in the parturient canal. Those cases which are not infected at that time generally suffer from infection sooner or later. This is so common that it is expected as a matter of course by the parents that the child will have a nasal discharge, and some parents are alarmed if the child does not, thinking perhaps that it is not like other children. This infection is not to be wondered at when we remember the perfect indifference with which the child puts everything into the nose or mouth. The regurgitation of the contents of the stomach, a part of which often comes through the nose, may be another source of infection. The child has relatively a much smaller passage through the nose than the adult, and this, together with its inability to clear the nose, increases the liability of the establishment of a purulent discharge. As these patients reach the age of puberty the increased breathing space in the nose and throat, and often the atrophy of the adenoid tissues, in the naso-pharynx, gives better drainage, and many of them recover. All, however, are not so fortunate, as the history of many cases of post-nasal catarrh will show.

In every case of acute rhinitis, from whatever cause, the accessory sinuses are involved. The inflammation results from an extension of the disease of the nasal mucous membrane by continuity of tissue, and it would be very hard to understand why this extension should not take place. The accessory sinuses are really a part of the nasal chambers and why, it may be asked, should an inflammation reach a certain arbitrary line and refuse to go farther?

A description of Rome which failed to mention the Forum would hardly be considered complete and a mention, at least, should be made of adenoids before leaving the subject of the predisposing and exciting causes of nasal empyema. This condition has been more closely studied than any other disease of the naso-pharynx, because it was early demonstrated that the presence of adenoid tissue in the naso-pharynx had a direct causal relation to ear disease in children. It is quite possible that the brilliant results obtained by the removal of adenoids may have led us to overlook other disease conditions which are of importance.

I wish at this time to call attention to a few erroneous beliefs which are prevalent in regard to adenoids. The first of these is the explanation that adenoids cause ear disease by obstruction to nasal respiration; every otologist of any experience knows that it is the location of the adenoid tissue around or in the Eustachian tube, and not the obstruction to nasal respiration, which produces the ear disease. Again, in regard to suppurating adenoids, that the uneven surface of an adenoid growth may retain pus or mucus, or that the removal of this tissue may lessen the amount of the discharge, or perhaps stop it entirely, no one denies; but that this suppuration originates in this tissue I do not believe: *a*, because the suppurative process would at once break down and destroy the soft tissue of an adenoid growth; *b*, because the discharge does not cease at once after the removal of the adenoid, as it undoubtedly would if this tissue were the seat of the suppuration. The explanation of the collection of pus on the surface of the adenoid tissue is that the situation of the growth high up in the vault of the pharynx, and often in the posterior nasal chamber, interferes with the drainage from the posterior ethmoid cells and the sphenoid sinus, and we have in addition to the adenoids an empyema of these cells.

Lastly, in regard to those cases of adenoids where the operation for their removal is a success, but the patient is not relieved. In this class of cases I have found the malformation of the middle turbinate, which I have described in this paper. Since I have been alive to the importance of this condition I have made it an invariable rule to examine for it every case of adenoids, and, when found, to give a guarded prognosis, *i. e.*, I have told the parents that the removal of the adenoids was the first step to be taken, and that the operation might improve the drainage to such a degree that nothing more would be required, but that there was disease of the nose which might still give trouble. I have found that the parents were much better pleased to know this before than to learn of it after they found that the operation was not a complete success.

Treatment: This will of course vary with the condition

found and the length of time which the disease may have been in existence. A large proportion of the acute cases, practically all of the lighter ones, will recover without any treatment or even with treatment which is distinctly injurious and it is only with the chronic or those acute cases which are very severe that we need concern ourselves at the present time. In general these cases may be said to have received inefficient rather than insufficient treatment. There are always two things to do in the management of these cases, one is to remove the discharge, and the other, which may be called the principal one, to improve the drainage to such a degree that the affected cavity will heal.

The removal of the secretion is not always the simple matter that it appears to be. The secretion is very often concealed by the middle turbinate and, to remove it, it is necessary to shrink the nasal mucous membrane and then to introduce a small canula between the middle turbinate and the nasal wall and wash out the accumulation. If the secretion is above the middle turbinate, it should be washed out by placing the canula in the upper meatus. The ordinary spraying of the nasal cavities is almost absolutely useless as a curative agent. After the secretion is removed it is often possible to see small masses of granulation tissue around the openings of the sinuses, which are the result of the inflammation within the cells. This granulation tissue may be the cause of the stenosis, or it may be the source of some secretion, and its removal by cauterization or by the curette is often all that is required to effect a cure. The thorough washing and the removal of granulation tissue should be given a fair trial before proceeding to more heroic measures; for, contrary to accepted belief, the nasal accessory sinuses have a strong predisposition to free themselves of inflammation and will often do so with but slight assistance. These are evidently the cases which recover spontaneously or by a change of climate, after a variable length of time. It is always well to keep these patients under observation for some time, because it is a well-known fact that a diseased accessory sinus may remain free from secretion for months and then relapse. This may be on account of focal disease

within the cell or on account of the anatomical construction of the cells or the parts surrounding them.

It is needless to add that these simple measures are not sufficient to cure those cases in which the drainage from the cells is interfered with by more permanent forms of obstruction. I shall not discuss the surgical treatment of nasal empyema in detail, except in so far as it relates to the malformation of the middle turbinate which I have described in this paper. **In general, surgical treatment of nasal empyema should aim to restore the nasal chambers to as near their natural condition as possible.** To help nature and not to improve her should be the end sought by the rhinologist. It should make no difference to us whether we can understand why the natural openings of the sinuses are at or near the top of the cavities or not. It is safe to assume that those openings performed their functions so long as they were free from obstruction and as a rule we shall not improve the situation by making others, unless there is extensive diseased tissue which must be removed. Even in these cases the natural openings should be freed from obstruction, because it is a noticeable fact that the artificial openings are not maintained for any length of time and the drainage from the cavities again takes place through the openings provided by nature. Again there is every reason to believe that focal disease of considerable extent, within the cell itself, will gradually heal if the natural drainage is made perfect. All focal disease will not disappear in this way, but it is always well to wait a time for nature in cases in which the urgency of the symptoms does not demand immediate operative measures. This advice applies to the further extensive operative measures after the obstruction to the free drainage from the cells has been removed. On the other hand it is useless to wait for atrophy to take place to improve the drainage in those cases in which the drainage from the cells is interfered with by a diseased or badly placed middle turbinate. The diseased middle turbinate and the diseased tissue around it should be removed without delay and the cells which are uncovered by the operation should be curetted. In the cases in which the middle turbinate is so closely

applied to the nasal wall that a probe can only with difficulty be inserted between them, enough of the middle turbinate should be removed to give free drainage, for even if atrophy does take place in ten or fifteen years and so improve the drainage, the post-nasal catarrh which is caused by the obstructed drainage has in that time usually done all the harm that was possible, and so far as the ears or the general health of the patient are concerned the cure has come too late. The same holds true in regard to the special malformation of the middle turbinate which I have described. It is necessary to remove enough of this to free the drainage. I have usually operated under cocaine with a small pair of Grünwald's forceps. It is not necessary to remove the whole of this part of the middle turbinate, but if there is a cell in the enlargement, as there usually is, it should be broken into. In a few cases the space available to operate in is so small that it is impossible to reach the part to be removed with the forceps or the middle turbinate may be so closely applied to the nasal wall that it is impossible to introduce a blade of the forceps between them. In these cases I have often succeeded in introducing a small saw and by sawing into the turbinate have made room for the blade of the forceps. Various expedients will readily suggest themselves to the operator.

If the assumption is correct that post-nasal catarrh is a symptom of nasal empyema and not a disease *per se*, then it is evident that it will be cured by the healing of the accessory sinus disease on which it depends. In attempting to prove this by a summary of cases which have been treated by improving the drainage from the accessory sinuses, I must of necessity rely upon the records of my own cases, as I am not aware that any cases have been reported which have been treated on these lines.

Within the last two years I have operated on 231 cases for the relief of chronic post-nasal catarrh; 123 of these cases had deafness of varying degree. Of these cases, 84 were operated on during the first year in which I did any work on these lines, and as a year or more has elapsed since they were treated we will analyze these cases for the reason

that the relapse of the disease can be better eliminated. Of the 84 cases,

78 had the enlargement of the posterior end of the middle turbinate.

23 of these cases had in addition diseases of other parts.

6 of the 84 cases had no enlargement of the posterior end of the middle turbinate, but did have obstruction to drainage around the anterior end.

The result of the operations to improve the drainage in the 84 cases was:

49 of the cases of post-nasal catarrh were permanently cured.

20 cases suffered relapse, but were finally cured.

7 were much benefited, but not cured.

8 were not benefited.

I have included in these records those cases which are known as post-nasal catarrh and have excluded those in which there was apparent extensive sinus disease. These cases were not included for the reason that there was no discharge into the naso-pharynx, but because they are usually classed as sinus disease, and not as post-nasal catarrh.

In regard to the effect upon the catarrhal inflammation of the middle ear which the relief of the post-nasal catarrh affords, in general, it may be said that it keeps pace with the condition of the naso-pharynx, although the improvement of the aural condition is much slower than the improvement of the condition of the naso-pharynx. It is evident that even after the source of infection has been removed, that some time may be necessary for the inflammatory process in the middle-ear cavity to subside. I have records of many cases in which the improvement in the hearing was immediate, but as a rule it is slow but fairly constant until the limit of improvement for each case is reached. A good rule for prognosis is this: by the cure of the post-nasal catarrh the hearing can be improved to nearly the hearing capacity which the patients enjoy under the most favorable conditions, *i. e.*, in regard to climate and freedom from post-nasal catarrh, and that this improvement can be held. It is

not claimed that the hearing is improved in all cases, for, of course, no one expects the hearing to be much improved if pathological changes of any extent have taken place in the middle-ear cavity. What is claimed is this, that those cases of chronic catarrhal inflammation of the middle ear which depend upon or are made worse by chronic catarrhal naso-pharyngitis can be prevented from growing worse, *i. e.*, freed from the danger of repeated exacerbations of the inflammatory process, by the treatment of the post-nasal catarrh on the lines which I have laid down. I have gone as fully into this subject of post-nasal catarrh as the limits of a journal article seem to permit. If I shall have directed attention to a new line of thought in regard to these cases, I shall be content.

In closing I wish to call the reader's attention to the following conclusions:

1. That the whole chain of catarrhal symptoms of the nose, the naso-pharynx, and of the ears is due to empyema of the nasal accessory sinuses.

2. That this empyema is the result of an infective inflammation of the accessory sinuses in which the drainage is insufficient.

3. That the malformation of the posterior end of the middle turbinate which I have described in this paper plays an important part in the establishing of the nasal empyema which causes post-nasal catarrh.

4. That chronic catarrhal inflammation of the middle ear may result from the catarrhal condition of the naso-pharynx, either by extension of the disease, by continuity of tissue, by the forcible blowing of the irritating secretion into the middle-ear cavity, or by closure of the Eustachian tube from involvement of the mucous membrane in or around its entrance.

5. That there is no evidence that chronic catarrhal inflammation of the middle ear is caused by obstruction to nasal respiration, unless the obstruction is associated with empyema of the accessory sinuses.

6. That those cases of chronic catarrhal inflammation of the middle ear which are caused or made worse by naso-pharyngitis cannot be cured until the nasal empyema which

causes the naso-pharyngitis is first cured, and that mechanical treatment directed to the ears is only palliative and does not free the patient from the danger of an acute exacerbation of the disease.

7. That many cases of nasal empyema may heal spontaneously under favorable conditions, and the more recent the case the more probable it is that this will occur.

8. That the accessory sinuses have a tendency to free themselves of inflammation, and that treatment should be directed to assist nature to this end.

9. That it is possible to cure practically every case of nasal empyema and therefore every case of naso-pharyngitis depending on it.

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THREE CASES OF DIABETIC MASTOIDITIS.

By Prof. E. P. FRIEDRICH, Kiel.

Translated and Abridged by Dr. Max Toeplitz, New York.

CASES of aural disease due to diabetes have not been frequently reported and then almost exclusively by Koerner, who has published four observations. This apparent rareness is contrasted by the fact, that during the course of the year 1898 I have seen and operated three cases of this kind, which are now fully reported, and confirm the views of Koerner on primary mastoiditis of diabetics.

The three cases are as follows:

Case 1.—Agent, aged fifty, well developed, has for years suffered from diabetes; the amount of sugar never exceeded one or two per cent. to the utmost and was easily suppressed by dieting.

On January 20, 1898, he was seized with ear- and headaches in the left side, associated with intense sensibility upon pressure over the mastoid apex. I found the surrounding parts of the outside of the ear unchanged, the mastoid apex quite painful upon pressure, the soft parts of the external auditory meatus swollen, but without secretion. The drum membrane was swollen, its surface softened, gray-red, except at a dark-red bullous bulging in its upper posterior half. Whispered voice: R normal; L=0; B. C. not decreased, and more accentuated towards the L E; low T. F. extremely, high T. F. less, decreased.

Upon paracentesis, a thin, sanguinolent fluid continued to escape during the following days, while the pain decreased. However, on January 26th, after cessation of discharge, it increased, in

order to decrease again after another paracentesis. The examination of the urine yielded with Fehling and Nylander a slight reduction only, owing to the strict sugar diet of the patient from the beginning of the present disease.

Until February 1st, the subjective symptoms had more and more abated, the sensibility upon pressure upon the mastoid had ceased, the external meatus was free; the second paracentesis, however, had been followed by persistent profuse suppuration. This condition remained unchanged for several weeks, until, towards the end of February, with dull headache and sleepless nights, the upper wall of the external auditory meatus was sinking, without external changes of the mastoid and without sensibility upon pressure. There was no sugar demonstrable. On February 28th, he was operated under chloroform. After exposing a discolored portion of the mastoid planum, from which chiselling was carried out, a widely branched system of pus containing osseous cells with a few granulations was laid bare. The bone was of dirty gray color, brittle and partially sequestrated, and was removed with spoon and rongeur as far back as the sinus, and downward to the extreme end of the mastoid apex. The antrum was spacious and filled with pus. With preservation of the tympanic ring and the ossicles, a wide opening was made into the recessus epitympanicus. The wound was left open.

On the day following the operation, the amount of sugar increased to 1.85 per cent., but disappeared after two days. The course of after-treatment was retarded by the detachment of a sequestrum from the tegmen tympani. The membrana tympani was cicatrized on March 8th, after entire cessation of suppuration after the operation; end of May, 1898, the wound closed; hearing faculty for conversation is preserved.

Case 2.—Sch.'s wife, aged forty-six, was admitted to the aural clinic at Leipzig on October 10, 1898.

The robust patient suffered for two weeks from an acute suppuration of the left ear, and for a few days from intense pain behind the ear. The left external meatus was filled with pus and the posterior wall bulging, the mastoid process sensible upon pressure, its soft parts slightly infiltrated. Whispered voice, L 16″. The examination of the urine yielded 5.85 per cent. of sugar.

A medium diabetic diet was instituted and the operation postponed. Since on the following day the swelling of the upper wall of the external meatus had so much increased as to obstruct

the entire lumen of the external meatus, and the infiltration over the mastoid had also considerably increased, the operation was performed on October 12th.

The narcosis was instituted with chloroform and continued with ether. After chiselling the cortex, slightly offensive pus emanated from a large bone cavity, with brittle discolored walls, communicating with the spacious antrum, which contained as small a number of granulations as the recessus epitympanicus and the middle ear. The posterior osseous wall of the external meatus was carious and contained here and there small cavities filled with pus. The mastoid process was abundant in cells, the bone brittle, gray-brown, without much pus in the cells. The radical operation was completed by the formation of Koerner's flap. The wound was sutured in the evening. T. 37.2° C.

October 13th.—2.42 per cent. of sugar. Vomiting, lack of appetite, thirst, refusal of solid food. Evening T. 37.2° C. Quantity of urine, 2200 *ccm.*

October 14th.—Change of dressing in the evening. The lower angle of wound was reddened and swollen, and pus discharged after removal of two sutures. Quantity of urine, 3100 *ccm.*

October 15th.—Since preceding night, deep, labored breathing, with the picture of beginning *diabetic coma* of dyspnœic character. Under slight dulness, the patient answered questions correctly with slow, scanning speech, but was otherwise completely apathetic. Pulse was small and accelerated. The somnolence increased during the day more and more, leading to complete loss of consciousness. The breathing became more labored, loud, and rapid. At noon, T. 36.0° C.; evening, 38.9° C. Quantity of urine, 800 *ccm.*

Death October 16th at 2 A.M.

The autopsy, made at the Pathological Institute of Leipzig, revealed: Intense œdema of the soft meninges and a markedly firm brain. From the suppurative wound at the left mastoid process *a phlegmon of the superficial cervical muscles extended down to the clavicle*, and laterally from the left lobe of the thyroid an encapsulated small abscess was found. The sinus and jugular were free. The lungs presented old pleuritic adhesions, extreme hyperæmia of both lower lobes, and œdema of the upper ones. The heart showed a dilatation of both ventricles and pale myocardium in fatty degeneration. Atrophy of pancreas, hypertrophied kidney with cortex in fatty degeneration, and dull swelling of spleen and liver completed the picture.

CASE 3.—Merchant, æt. forty-two, was admitted on December 18, 1898, to the aural clinic at Leipzig.

Patient, an inveterate drinker, was seized two years ago with suppuration from the left ear, which persisted ever since with varying improvements and aggravations. On December 11, 1898, he suddenly became worse, with simultaneous pain and swelling behind the ear, forcing him to call for admission to the hospital. The robust patient had a small, irregular, unequal pulse. The urine contained albumen and five per cent. of sugar, as was accidentally found.

The *left* maxillo-mastoid fossa was filled out by an elastic, uniform swelling, which extended upward over the mastoid process to the temporal line and down the posterior edge of the sterno-cleido-mastoid muscle, with reddened skin and deep-seated fluctuation. The walls of the external meatus were diffusely swollen, the membrana tympani invisible, and profuse non-offensive purulent discharge issued from the ear. The radical operation was not performed owing to the high percentage of sugar and the weak heart action.

On December 19th, the abscess was incised, under local anæsthesia, with an ether spray. The incision was made from the mastoid apex forward and downward, and reached the abscess only very far inward, when a large quantity of pus escaped. The abscess cavity extended far forward and downward; the probe met upward rough bone at the mastoid apex. During the after-treatment the daily quantity of sugar varied between ½ and 1¼ per cent., and the quantity of urine between 1000 and 1500 *ccm*, and, one day only, 1800 *ccm*. The action of the heart continued to be weak, the urine contained much albumen, and, in addition, profuse diarrhœa appeared.

On December 28th the patient left the hospital; the wound discharged pus profusely during the following weeks, the soft tissues remained infiltrated even after the incision was closed, and the otorrhœa ceased at times.

The serious aspect of aural disease in diabetics is the rapid extension of osseous caries, which should be early and extensively removed. During the operation it is found, as a rule, that the subjective and objective symptoms of the patient are out of proportion to the extreme extent of the disease. Early operations are often followed by difficulties based upon the nature of diabetes mellitus.

The danger from operating on diabetics consists in the subsequent appearance of sepsis and coma. Both complications depend upon the amount of sugar and the acidity of the urine, which, when found together to a high degree, form a contra-indication of the operation.

The danger from sepsis is greater in aural operations which present septic wounds and often give rise to mixed infections, owing to the communication of the pus from the diseased osseous parts with the external meatus through the middle ear. The above reported second case illustrates the rapid development of a burrowing abscess along the superficial cervical fascia.

The appearance of diabetic coma is not due to the operation or to shock, but to the narcosis. In some of Becker's cases it did not set in until the second day after the operation. The kind of narcotic used is not of so much importance as the metabolic change thereby produced through the increased acidity. This is well illustrated by my second case, while the first one presented an increased amount of sugar on the day following the operation.

In all cases in which the aural disease requires an early operation, the general health of the patient and the condition of his circulatory apparatus, lungs, and kidneys should be considered. In the third case, the nephritis associated with myocarditis and arterio-sclerosis forbade the operation in narcosis.

In some cases it is difficult to determine the proper treatment, since a serious aural disease, which urgently requires an operation, is complicated with a constitutional disorder which may eventually lead to a fatal issue.

The operation of the robust woman of our second case was a mistake, and its unfavorable course may serve as a warning to be cautious in future cases, but not restrict us from all operations, since simple incisions, as in the third case, are in themselves not of great importance.

In touching briefly upon the question, how in future similar cases the fatal issue may be avoided, the omission of the narcosis as the greatest danger would be most important in the treatment of diabetic mastoiditis. It is to

be regretted that the results of local anæsthesia are not as yet good enough to consider its use except in severe cases.

Naunyn's suggestion of the administration of bicarbonate of sodium as a prophylactic in operations on diabetics, in addition to a regulated diet before and after the narcosis, in order to avoid the intoxication with acids and the danger of the appearance of coma, is to be commended.

SHARPLY CIRCUMSCRIBED SOUND-DEFECTS IN THE HEARING-FIELDS OF CERTAIN DEAF-MUTES.

By Dr. A. SCHWENDT,

PRIVAT-DOCENT IN BASEL.

Translated and Abridged by J. Guttman, M.D.

Case I.

THE seven-year-old deaf-mute, Albert T., became deaf at the age of seven months in consequence of an attack of meningitis. The right eye is completely blind.

Both drum membranes are about normal.

The patient is absolutely deaf in his left ear; on the right side, however, there is a fair amount of hearing, especially for the middle octaves. The right ear can quite well distinguish all vowels.

The lower limit of his hearing power lies at small c, the upper at h^4.

The most peculiar feature in this case is a sharply defined deafness for the note f^3, whereas c^3 and g^3 are clearly heard. This tone-defect is best demonstrated with the aid of Koenig's high tuning-forks c^5–f^5, which are accurate instruments and produce very high notes.

We cannot as yet decide whether the note f^5 could be heard or not, if it were produced with greater intensity.

If these notes are produced with the newly improved Edelmann's Galton whistle, we find that the mark 13.6, which corresponds to the note f^5, can be heard only in the immediate vicinity of the ear. It follows that so close by

the ear he perceives either the harmonics of f^4 or only a tactile sensation.

This deaf-mute can hear at a greater distance the notes which are produced by the prolongation or shortening of the whistle.

Case II.

The nine-year-old deaf-mute, Charles S., became deaf in his third year in consequence of influenza. He is absolutely deaf for the upper half of the scale. His hearing power is about equal in both ears. The lower limit of his hearing lies in both ears at contra C, the upper limit right at f^1, in the left at a^1.

He cannot distinguish vowels but he can hear them if they are shouted.

The only consonant which this deaf-mute can recognize is the lingual R.

His field of hearing, *e. g.*, the duration of his perception of the note c, as expressed in percentages of the normal, is:

	Right Ear.	Left Ear.
c^5	0	0
c^4	0	0
c^3	0	0
c^2	0	0
a^1	10 %	10 %
c^1	30 %	70 %
c	33 %	72 %
C	34 %	80 %
C^1	50 %	80 %

Both cases demonstrate the sharply limited defects of hearing power for certain notes or for certain parts of the sound scale, which is often met with in cases of deaf-mutes, or in persons who have some affection of hearing.

This second case contrasts sharply with the following case of deafmutism.

Case III.

A twenty-one-year-old girl became deaf in consequence of an attack of meningitis and is completely deaf in her right

ear. In the left, the upper limit lies immediately above g^3. She possesses comparatively good hearing power for the notes below g^3; for the notes above that point she is completely deaf.

Her duration of hearing as expressed in percentages of the normal is as follows:

	Left Ear.
c^5	0
c^4	0
c^3	0
b^2 a^2 h^2	a high limit
c^2	50 %
c^1	95 %
c	65 %
C	60 %
C–1	60 %
D–2	low limit

The upper limit varies between $b,^1$ a^1 and h^1 according to her daily disposition; when she becomes tired, the duration of tone perception as well as the acuteness of hearing of speech is diminished. In her ordinary disposition she can hear in the immediate vicinity of the ear moderately loud conversation; if the conversation is too loud she is annoyed by it and says that she cannot understand it as well. In contrast to our deaf-mute, Charles S., she has a comparatively good hearing power for the octave g^1–g^2.

Charles S., can hear of the consonants only the lingual R; the girl on the other hand can hear all consonants with the exception of S. Our deaf-mute Charles S. constitutes a contrast also to the two deaf-mutes demonstrated by Bezold at the Naturforscher-Versammlung at Munich. These two had a good hearing power for low notes, but only a very short duration of hearing for the notes g^1–g^2. In spite of this they could hear speech quite well, although for the hearing of speech a much longer duration of hearing for g^1–g^2 is required. The perception of the low notes had in this case evidently a favorable influence upon the perception of speech.

Bezold explains this phenomenon by the aid of the Helmholtz–Hensen theory as modified by Ebbinghaus. According to Ebbinghaus, the fibres of the membrana basilaris which are intended for the low notes come into oscillation not only by the original note to which they are tuned, but also by the harmonics through formation of nodules. In this way only can we explain the peculiar phenomenon which we observed in the patients demonstrated by Bezold.

MULTIPLE RAREFACTION, "SPONGIOSIRUNG," OF THE LABYRINTH-CAPSULE FOUND AT THE AUTOPSY OF A CASE OF PROGRESSIVE DEAFNESS.

By Prof. F. SIEBENMANN, Bâle.

(*With eight illustrations on Plates I.–VI. of Vol. XXXIV., No 4, German Edition.*)

Translated by Dr. Arnold Knapp.

ONE of the two cases described by my pupil, Edward Hartmann, in vol. xxx., p. 1, of the *Zeitschrift für Ohrenheilkunde*, was of especial interest because v. Tröltsch had made the diagnosis of nervous deafness from the functional examination (markedly diminished bone-conduction). At the autopsy and microscopic examination bony ankylosis of both stapedii and an extensive rarefaction of the bony labyrinth-capsule were found present, and on again looking over the specimens, I discovered a considerable exostosis in the lower parts of both scalæ. Nerve bundles and ganglia of the auditory nerve and Corti's organ seemed normal. The bony canals of the tractus ganglionaris and of the tractus foraminulentus were contracted.

Another case of deafness where I had made a functional examination, and later a post-mortem investigation of both auditory organs, enables me to conclude that extensive rarefaction of the labyrinth-capsule is sufficient, independent of an involvement of the bony nerve canals, to produce a decided diminution of bone-conduction. It shows that the same process, according to the localization, may produce a bony stapes-ankylosis or progressive nerve deafness with correspondingly different functional findings. This micro-

scopic condition is interesting from another view point and is, in fact, unique, as in both labyrinth-capsules—in the cochlea as well as in the semicircular canals—a large number of isolated foci existed. These specimens can decide in a definite manner the previously unsolved question on the origin and further development of these pathological rarefying processes.

K. S., female, fifty-two years old, was admitted to the hospital on December 30, 1896. Except for an eczema of the arms, patient has always been well. One month ago an attack of sciatica, then frequent chills, nausea, and fever. The clinical diagnosis of endocarditis ulcerosa was made. She died on January 5, 1897, and at the autopsy the following conditions were found: Endocarditis ulcerosa, myocarditis, miliary abscesses of pia and cortex, hemorrhagic infarct of the spleen, infarct and abscesses of the kidneys, miliary abscesses in the submucosa of the stomach and intestines, abscess of the left thyroid gland, parotitis, embolic hemorrhages of the larynx and trachea.

I was able to examine the patient on December 31st. She stated that she had gradually become deaf in the last few years. There had been no otorrhœa, but frequent pain in the ears. In recent years attacks of vertigo without vomiting occurred, which would necessitate lying down. As regards heredity, the father had been very deaf; the other relatives, however, had normal hearing.

The examination, on December 31st, revealed normal drum membranes. Whisper was heard right at 4 *cm*; left, 150 *cm*. Fork a′ was lateralized from the vertex to the left (the better) ear, and was shortened ten seconds. Rinné a′ positive on both sides, right approximately of normal duration (about twenty-five seconds); left not crossed (not carried over to the right ear); A perceived on both sides even on slight impulse. Owing to the critical condition of the patient the examination could not be prolonged; the determination of E Rinné left, and the upper and lower tone limits had to be given up. The diagnosis of bilateral progressive nerve deafness seemed justified.

Twenty-four hours after death we examined the two temporal bones, and macroscopically the external and middle ears seemed normal. The labyrinths were freed, and the superior semicircular canal opened; they were placed in

formol, dehydrated, decalcified in hydrochloric acid, imbedded in celloidin, and finally cut into about three hundred vertical sections, in the plane of the superior canal. Every tenth, and, in the region of the oval window, every fifth section were stained with eosin-hematoxylin; later, control sections were stained with neutral carmin, picrocarmin, hematoxylin carmin, and according to Weigert-Pal. Very beautiful pictures were obtained by overstaining with carmin, then hematoxylin, and decolorizing with a watery solution of picric acid plus a trace of hydrochloric acid (one-half per cent.).

The following interesting conditions were found and are reconstructed into Figs. 1 and 4:

a. Right labyrinth (the lateral extremity, *i. e.*, the vertex of the lateral and posterior canals, is wanting): the nerve and membranous labyrinths seemed normal. In each of the two bony semicircular canals, partly preserved in the specimen, there was a focus of rarefaction, a third focus was found at the oval window, a fourth at the stapes plate, a fifth and sixth at the cochlear capsule (see Fig. 1).

The first focus extends from the canalis subarcuatus to the inner (concave) wall of the upper semicircular canal in its vertical portion and is adjacent to the endosteum; it partly limits the ampullar extremity in front but does not invade the ampulla itself. To the medial side of the superior canal the first area terminates abruptly, on the lateral side it extends farther than is seen in the specimen. Posteriorly, it connects by means of prolongations with the second area which is situated on the ampullated end of the posterior canal.

The third area surrounds the oval window above, below, and in front so that the posterior half of the lower margin and the posterior portion of the window remain free. A freely vascularized periosteum is situated beneath the mucous membrane of the window niche as far as the bony changes extend.[1] On the inner side this area extends over

[1] The fact first advanced by Schwartze and verified by various authors, that the labyrinthine wall of the middle ear especially in peracute cases of sclerosis shines reddish through the drum membrane, is probably due to unusually increased vascularity of these bony parts and to the change of the thin mucosa into vascular thick periosteum. I have observed this condition frequently in youthful individuals, in purely progressive nervous deafness and not alone in stapes-ankylosis.

and in front of the window so that it directly forms a portion of the facial canal wall, and of the nervous utriculo-ampullaris of the pyramid and of the crista vestibuli; a part of the bony cochlear wall is involved, especially the wall opposite to the vestibule, as well as the vestibular and the tympanic scala and the portion corresponding to the area between the basal and middle turns. The upper fourth or third only of the labyrinth wall situated between the oval and round windows shows rarefaction. As to the rarefaction of the window margin it is converted into rarefied or osteoid tissue, except a part of the lower circumference of the cartilage; the annular ligament is decidedly diminished. On the vestibular side the window margin is replaced by a rarefied wall which above and in front surrounds the edge of the stapes plate on both sides; it resembles in thickness and structure true vascular periosteum. The more recent tissue is found in the superficial layers of this osteophyte. It is osteoid in character and takes on a deep blue stain. The free surface under the periosteum is uneven, rough with coral-like projections, and the intervening spaces contain a homogeneous pinkish staining mass similar to that in the deeper layers of the periosteum. The window niche is somewhat contracted.

The stapes plate is not thickened, but its vestibular cartilaginous covering is converted into bone in the middle (fourth area).

The fifth and sixth areas are in the cochlea. Both are situated deep and do not approach the tympanic mucous membranes. The fifth has a flat, sausage-like form and is situated in the tympanic wall of the tympanic scale at the basal turn (at its lower and inner part, see Figs. 2 and 3); it forms toward the vestibule the lower and toward the cupola the outer cochlear wall and also the lower margin of the int. audit. meatus, somewhat altered by osteophytic proliferation. The endosteum of the cochlea does not show any thickening at this point, but in the places where the spiral ligament is covered with rarefied bone there are several osteoid homogeneous plates or bone corpuscles intensely stained with hematoxylin. The two small extremities of the area do not reach the cochlear canal. The bone is normal

in the area of the tractus foraminulentus; the above-described changes in the wall of the meatus are situated to its outer side.

The sixth area (Fig. 3) is likewise situated at the limit between the upper and lower part of the basal turn; it covers the latter in the vestibular scale, approaches the middle turn, and extends for a short space in the direction of the apex without invading the cochlear lumen.

The only connection between the areas is a slight one between the first and second. The remaining four are isolated.

A description of the structure of the spongiosa and its varying condition at different places will be given later after the left temporal bone has been described.

b. Left temporal bone (all of the semicircular canals are well preserved in the specimens); see Fig. 4:

The nerve and bony labyrinth are normal. The bony canals of all three circular canals are surrounded by an area of rarefaction (see Fig. 5); the ampullæ are free. The lateral canal is the least affected where the middle of the crus simplex shows the above-described changes on the upper surface. The posterior canal is principally affected, especially in its entire length. Of the superior canal (Fig. 6) the ampullated extremity is chiefly involved, and especially at its inner surface and its concave (inner) edge.

The fourth focus is situated about the oval window, and has about the same shape as area three of the right labyrinth, though it is somewhat more extensive. At the posterior upper window margin the bone is rarefied but the cartilaginous margin is unaffected; the posterior lower part is normal both as to bone and to cartilage. Otherwise the cartilage is everywhere replaced by spongiosa. At one place (see Fig. 7) the ligamentum annulare and the lower stapes margin are converted into spongioid bone, which without interruption passes over into that of the lower window margin (bony stapes-ankylosis). At other places, the stapes presents its normal cartilaginous margin. The upper stapes margin is dislocated externally, in its anterior part by the osteophytic hypertrophies and the consecutive narrowing of the window

(see Fig. 7). The pelvis ovalis is deepened and narrowed by the proliferation of bone. The focus extends into the depth between the vestibule and the middle turn of the cochlea, limiting the latter and just touching the apex extending to the fundus meatus without involving the endosteum of the basal turn or the canal of the modiolus. The walls of the facial canal, vestibulum, and of the utriculo-ampullar branch are rarefied similar to the right side. Further foci (like five and six of the right temporal bone) are wanting.

The pathological diagnosis is therefore: *on both sides, areas of rarefaction in the bony capsule of the circular canals, of the vestibule and cochlea. Formation of osteophytes on the vestibula and tympanic surface of the oval window margin. Commencing ossification in the cartilaginous covering of the stapes. Additionally, on the left side commencing ossification of the annular ligament (incomplete stapes-ankylosis).*

The specimen was well preserved and fixed, and was examined in serial sections, so we were able to investigate, 1st, the various developmental stages of this "spongiosa" formation, and, 2d, the origin or starting-point of the process. As to the first question, we can say that the first stage consists of a change in the Haversian canals, inasmuch as they lose the relation of the innermost layer to hematoxylin and carmin, then they enlarge on the labyrinthine side in funnel-like spaces to large lymph cavities by lacumar resorption; multinuclear giant-cells are often present. The round and star-shaped cells of the perivascular lymph spaces multiply, and connected by thin processes to form a loose network, they fill in the space between the bony wall of the cavities and the delicately walled vessels. In a further stage, at those places where the resorption process has ceased, the cells of the peripheric zone are attached as broad shallow osteoblasts to the wall of the cavities, forming a gradually thickening concentric area of decalcified tissue which stains deep red with carmin, violet, and in places, dark blue with hematoxylin-eosin. An occlusion of the space down to the vascular lumen does not occur; the stronger refracting cement-line (Pommer) between the unchanged original and adjacent new bone remains well marked. The two latter areas are

distinctly different at this early period, inasmuch as, apart from the staining differences, the new-formed osteoid zone is marked by less clearness and by numerous coarse *irregular* bone corpuscles which are in part enlarged, and often possess numerous abnormal distinct prolongations as well as one or two very deeply stained nuclei in a light area. *All the Haversian canals and spaces in this affected area are changed by this resorptive and appositional process, and in addition all cartilage containing introglobular spaces* (Manasse-Gegenbauer) *are dissolved and replaced by new-formed osseous tissue.* The bone about the cartilaginous part of the labyrinth window—later also of the stapes—is absorbed by large penetrating blood-vessels, and replaced as above described by the spongioid tissue. Bright osteoid plates arranged in chains, at first free from bone corpuscles, stained blue with hematoxylin, appear isolated in the annular ligament; after the cartilage of the stapes and window margin have almost been brought to touch by proliferation of the bony understructure, communicating bridges are formed over this narrow cleft. These are arranged radially, more in a horizontal than vertical plane, and are attached at the innermost point of the window margin, and run together, changed into spongiosa communicating among each other and with the cartilaginous margin or with the bone which takes the latter's place.

In the later periods, the intervening walls of the medullary spaces become thicker and the spaces smaller. The bone tissue stains with hematoxylin-eosin a bright pale red color, and loses, except at the inner zone, its relation for carmin. It is an important fact that the new bone gradually assumes a lamellar structure; the bone corpuscles are arranged concentrically and the nucleus atrophies. The medullary spaces become poor in cells and vessels but richer in fibrous connective tissue running parallel to the axis of the canal. The limiting lines grow gradually less distinct and disappear (as Hanau has described in the epiphyses of the ribs, see Mader, "On Inflammatory Hyperostosis and Periostosis of the Ribs in Pleurisy," *Archiv f. Entwickelungsmechanik*, vol. vi., No. 4). Abnormally active regeneration

and resorption of bone are seen more distinct at places associated with pale red or pale blue zones outlined by sharp lines about the Haversian canals, without the latter undergoing a distension.

The characteristics of the two stages correspond exactly to the pictures which I have observed (in seven similarly diseased temporal bones) either, at the very beginning, as an area of pin-head size, or, after thirty years, changes extending over the entire labyrinthine capsule. I agree with the views of Bezold on the histological conditions.

There is another reason to show that the decalcified carmin zone is new-formed bone, as Pommer and recently Hanau have shown. The osteophytic tissue possesses in every relation the same microscopic structure, and the same in reactions as the carmin zone in the deeper area. In the latter the process of resorption and placing together can be beautifully followed in those places where the advancing resorption line has reached a cartilaginous introglobular space, and a fundamentally different structure is deposited. Small deviations from this order are occasionally seen; old bone may stain a light blue with eosin-hematoxylin instead of red; in the younger parts of bone the bone corpuscles are frequently not unusually dilated or numerous, etc.

After we had verified our conclusions on these histological changes by examining other specimens of osseous tissue, we went over all of the sections again to investigate the local origin of this process. This had the very interesting result that this rarefying process does not emanate from the periosteum (which Bezold at least does not exclude). Nor does it arise originally in the labyrinth capsule (Politzer), but that the *oldest parts occur at the limit between the endochondral primarily formed labyrinth-capsules and to the connective-tissue bone secondarily deposited from the periosteum* (probably in the latter itself). For we find the most recent richly nucleated areas stained deeply with hematoxylin-eosin directly at the endosteum of the labyrinth-capsule; the oldest are situated in the centre of the circular canals, about the entering large bony vessels. In the spongioid focus occupying the space between the oval window, vesti-

bule, cochlea, and facial nerve, the most recent portions are situated at the lower window margin in the superficial layer of the ring of osteophytes on the vestibular surface of the oval window-ledge near the canal for the utriculo-ampullar nerve, while the older parts occupy the centre. The latter is situated somewhat in front and over the anterior margin of the oval window and forms, as other authors have found, the place of predilection for this affection. It is without doubt due to this condition that the osteophytic wall usually is higher in front than in the back, and that the upper margin of the window is more extensively affected than the lower. A more pronounced development of the wall at the posterior margin occurred in Politzer's fifth case. In Bezold's third case the changes in the bone were pronounced posteriorly and below, but no wall was present.

Area five in the right temporal bone is relatively old; the most recent spongiosa is (as in the area described in Bezold's case two) directed to the cochlea, the oldest is situated near the porus acusticus. The entire area six is more recent; one part is, however, older; it is pale red, lamellated, and poor in nuclei, separated by no limiting line from the healthy bone, and situated farthest away from the cochlea and directed to the tip of the petrous bone. There were no exostoses at the lower part of the promontory, as Habermann has described, or in the basal turn, as Politzer saw projecting into the scala tympani of the basal turn (cases five and six), and as were present in the two temporal bones of my collection published by E. Hartmann. No changes were found in the canalis ganglionaris, thus differing from the two latter mentioned temporal bones and case five of Politzer.

It is noteworthy that the spongioid spaces are nowhere so large that it is permissible to speak of an osteo-porotic process. In most of the cases there is an active apposition; though there are numerous places where only resorptive processes are visible, especially in the neighborhood of the nerve channels and beneath the endosteum of the labyrinth, *so that the endolymphatic fluid is separated only by a connective-tissue and frequently very thin septum from the large lymph spaces of*

the spongioid area. Broad perivascular spaces connect the labyrinth with the porus acusticus and with the tympanic cavity.

An interesting question naturally presents itself: Why does the labyrinth-capsule at so late a period show this tendency to convert its compact, ivory-like bone into loose spongiosa? The process is all the more striking as similar changes have not been observed or suspected from the history in any other part of the skeleton. An explanation for this peculiar process is, according to our idea, to be found in the fact that the normal *labyrinth-capsule* remains throughout life unusually *rich in remnants of primary cartilage.* This occurs in the form of small and large deposits near the labyrinth spaces and is noticeable in sections by the deeper staining with eosin-hematoxylin. This staining does not affect the cartilaginous remnants alone, but the entire inner zone of the labyrinth-capsule and the outer zone, especially the area opposed to the periosteum, are stained bright red. The introglobular spaces surrounded by irregularly shaped walls are most frequent in the neighborhood of the posterior half of the oval windows and in the basal end of the upper, also of the lower cochlea wall—in other words, *in those regions which serve as places of predilection for the spongiose formation.* (We have recently accidentally encountered an almost hemp-seed-sized piece of true hyaline uncalcified cartilage in the labyrinth-capsule of an old person. This was situated between the posterior edge of the oval window and cochlea, permeated with large oval cartilage cells. It lacked the peculiar superficial structure of the introglobular spaces, and took on a much deeper stain than the latter.) The semicircular canals, the margin of the round window, and the modiolus contain less cartilage and are consequently less prone to undergo the osseous change.

Similar to the cartilage remnants which are situated like discs between the epiphysis and diaphysis of the long bones and the parts of the skeleton derived from the procartilaginous third and partly second branchial clefts which commence to ossify in juvenile years, and like many cartilaginous

tendon insertions which later change to cancellous bone, we observe a process similar, though exceptional, occurring in the bony labyrinth, which terminates with the disappearance of the cartilage at the window margin and in the intro-globular spaces. At the same time, the compact bone changes on to the type of connective-tissue bone with the formation of a fibrous medulla and periosteal deposits, the latter especially about the labyrinth window, in the stapes plate, in the oval window niche, and in the lower part of the cochlear spiral.

If I understand Koellicker (*Handbuch der Gewebelehre*, vol. i., p. 346) correctly, a similar process has been observed by him and by Strelzoff during the growth of the scapula and of the long bones in certain places and considered to be normal; except that there the resorptive process has predominated over the displacement of bone formed from cartilage by connective tissue. I do not from this fact wish to call the rarefaction of the labyrinth-capsule an osteitis, but rather to regard it as the final stage of a developmental process which normally does not occur in the petrous bone, though it is the rule in other bones, though not in the same form and at the same time.

In all the long and flat bones of the skeleton there is a continuous loss and regeneration after birth, so that the bone continues to grow without changing its external shape. In the labyrinth-capsule, however, there is an exception, as the size is attained at birth and a later decrease or increase, as far as the examination of the bone shows, takes place only in small limits. This probably is the reason that so many cartilage remnants are contained in the labyrinth-capsule to an old age, while in the other bones which grow, they generally disappear early.

It appears that a compact limitation of the labyrinth-capsule is important for the function of the organ therein contained, as this is pronounced in all higher developed animals. An active regenerative process as in the other bones would carry with it a disturbance in the position of the nutritive vessels and also of the cochlear canal; a more extensive blood supply from the side of the labyrinth and a closer connection

between the intralabyrinthine vessels with those of the bone capsule would be required. We know, however, that Hyrtle's views on the closed-in system of labyrinth vessels correspond practically to the actual state (see Siebenmann, *Die Blutgefässe im Labyrinth des menschlichen Ohres*, 1894) and that the blood-vessels of the endosteum communicate in only a few places through narrow capillaries with the blood current in the bone. The lymph circulating in the Haversian canals between the blood-vessels and bony wall is shut off from the general labyrinthine lymph space. The high importance from a functional standpoint of such a separation of the blood and lymph distribution is seen from the following condition found at autopsy in connection with the vessels of the hearing tests: bilaterally, except a delicate osteoid bridge at the left annular ligament, there are no changes in the middle ear, but there is an extensive rarefaction of the cochlea and semicircular canals and bone-conduction is very much reduced.

We were unfortunately unable to make a satisfactory functional examination in our case; hence the lower-tone limit could not be determined and the not very marked immobilization of the stapes was not diagnosticated in the living. We have no explanation for the decidedly reduced bone-conduction other than the changes just described of the labyrinth-capsule. This supposition gains force as there were no other anomalies in the labyrinth, and it is made almost certain by the fact that the hardness of hearing was found not on the side of the stapes-ankylosis but in that ear where the spongiosa formation had progressed farthest.[1]

In both cases of Bezold where stapes-ankylosis was found at autopsy, the spongiosa also extended to the endosteum. The fact that bone-conduction was here not shortened but rather prolonged appears to contradict my explanation but is due to, first, the thickness and breadth of the spongiose bridges in the annular ligament—*i. e.*, the bony stapes-ankylosis has reached such a marked degree in both cases,

[1] As abnormal rarefaction of bones is observed in the late forms of syphilitic disease, it would be well in future to examine more carefully the labyrinth-capsule at the autopsy of the syphilitic deaf. The well-known observations of Moos and Steinbrügge might be explained in this manner.

and, secondly, the rarefaction in one case has extended beyond the oval window and in the other not at all. Moreover, though bone-conduction was prolonged in both cases, it cannot be excluded that, at the time of the functional examination, the labyrinth function had already become affected. This is even more than probable when we compare the degree of prolonged bone-conduction in these cases with the prolongation observed in cases of stapes fixation produced artificially or by depression of the drum. In affections of the Eustachian tube [1] and in the indirect traumatic ruptures of the drum membrane [2] a prolongation of fifteen or nineteen seconds (measured with a Bezold-Katsch fork) on the affected ear, and ability to hear whisper in 20-60 *cm* were present, while in Bezold's cases of ankylosis similar changes in bone-conduction meant only hearing whisper in 6 *cm.* I have several examples, verified by Rinné's test, to show that in a pure middle-ear trouble (total closure) of young people the hearing distance for whisper may be one metre or more, in cases where Schwabach's test shows a prolongation of eighteen to twenty seconds. These two cases of Bezold's, therefore, support my view that rarefaction of the labyrinth capsule, if it extends to the endosteum, of itself affects the labyrinth function and causes a relative diminution of bone-conduction. I should just like to mention that I had reached a similar conclusion (*Z. f. O.*, xxii., p. 315) by clinical and experimental means, and proposed the statement that the labyrinth is always implicated in progressive bony stapes fixation, even in the supposedly pure cases—*i. e.*, even in complete presence of the characteristic functional symptom-complex.

It is not necessary here to discuss how, by the overlapping of the results of the functional examination of stapes-ankylosis and of the nervous progressive deafness, finally an atypical picture resembling the latter is produced, as v. Tröltsch has intimated. It suffices to say that a large proportion of such cases belong in this list which Bezold,

[1] Siebenmann, "Horprüfungsresultate bei reinem Tubencatarrh," *Z. f. O.*, xxx., p. 308.

[2] Nother's "Traumatische Perforationen des Trommelfells," *Z. f. O.*, xxxiii., p. 19.

unable to classify among the middle-ear or labyrinth affections, placed in a special class under "Dysacusis." I should like to emphasize the importance of the determination of the lower-tone limit in general as a differential means of excluding the pure nervous deafness from the class of such combination pictures.

We have seen that rarefaction of the cochlea capsule, wherever it occurs, affects the function of the nervous terminal organ. In which way does such a disturbance occur and how can it be explained? The solution probably deals with *changes* in *pressure* and *density* which the labyrinth fluid suffers by influence of the spongioid spaces extending to the labyrinth fluid. Chemical changes surely take a part and assist in increasing the nutritive change in the delicate elements of Corti's organ. The extensive and in part very thin diffusion surfaces which in some spaces alone separate the enormous lymph spaces of the new-formed spongiosa from the perilymph of the labyrinth in the form of very thin membranes, increase greatly the number of existing lymph passages of the perilymphatic duct and of the perivascular spaces (however, only indirectly affected). While this was the chief communication to the interior of the skull or to the posterior cranial cavity, the labyrinth fluid now enters upon new relations to the peripheric lymph and vessels subjected to other pressure conditions. An alteration in the conditions of diffusion may not be the only change; as can be seen at several places in my specimens, the septum (reduced to a connective-tissue membrane) between the two lymph systems may also be absorbed; perforations with sudden changes in intralabyrinthine pressure and position may occur, and of such a kind that a reproduction of the former condition is not again possible. This is the only explanation for the loss of hearing and diminished bone-conduction, as the Corti's cells, stria vascularis, nerve, and labyrinth windows showed no marked changes microscopically. It can thus also be explained that just in this case vertigo with diminished hearing appeared in attacks, and that the hearing slowly or incompletely or never was brought back to the previous condition. Ménière's vertigo,

the morbus Ménière in the mild form and in the severe form with vomiting, nystagmus and excessive vertigo in apoplectic attacks, is not sufficiently explained[1]; we think, however, that thereby a solution has been found, and are further convinced as we know that these patients usually present the other symptoms of so-called sclerosis. The peculiar subjective noises, as thunder, thumping, shooting, from which the patients suffer terribly, can be without difficulty referred to these perforations.

I will only casually state here that variations of density of the labyrinth fluid must be associated with alterations of sound-conduction. A diminution of labyrinthine pressure is possible in our cases; this would of itself as direct cause explain a diminished sound-conduction through the labyrinth fluid and the shortening of cranial bone-conduction. We must by all means consider all of these factors if we wish to explain the remarkable fact that spongiosa formation of the labyrinth capsule with stapes-ankylosis produces in one case a prolongation, in another a shortening, of bone-conduction. In a case since examined at autopsy, a period preceded the stage of shortened and finally absent bone-conduction where Bezold's trias of stapes-ankylosis was well developed.

The question whether we should in future retain the expression sclerosis of the ear is to be answered negatively. The juvenile form of this symptom-complex is not a condensing process, but, as far as the bone is concerned, just the opposite. Progressive nervous deafness of older age depends, as we have shown, in most of the cases on quite different changes; a fact important both for the diagnosis and treatment. In future we will classify such cases of early or late appearing progressive deafness with aid of the other factors, important for diagnosis, according to the result of functional examination in *nervous deafness* or *dysacusis* (Bezold) or *stapes-ankylosis* with the addition: rarefaction, "spongiosirung," of the labyrinth-capsule.

[1] The implication of the canal pro nervo utriculo-ampullaris in the process of spongiose formation does not change the nerve or its connective-tissue sheath. The changes found in the bony circular canals, as the ampullæ are unaffected, can only in the above-described manner and way contribute to cause the attacks of vertigo.

In regard to the treatment, remembering the results of the autopsy it can be positively stated that local medication, treatment of nose and throat, injections of medicines into the tubes, massage of the drum, myringectomy, tenotomy, and stapedectomy, excision of oval window, etc., as well as the use of potassium iodide, thyroidin, and pilocarpin, are of no avail and may aggravate the condition by their irritating action. Hence such treatment must be abstained from where the diagnosis of rarefaction of the labyrinth capsule is made. The fact that catheterization not rarely produces an improvement of hearing in progressive nervous deafness, if only transient, can be explained because in spongiose formation of the region of the cochlea tips the lumen of the bony tube is somewhat narrowed (only recognized, microscopically, by thickening of the periosteum). Of the internal means, phosphorus alone seems to me to promise anything. This, according to recent investigations (Mirwa and Stötzner, *Jahrbuch für Kinderheilkunde*, vol. xlvii.), in rational administration is able, at least in the long bones, to prevent the formation of the (normal) spongiosa and to favor the formation of compact bone. I usually prescribe an oily solution or Kassowitz's emulsion, 0.01 %, and give 10-20, later 30-40 *ccm* daily. If the stomach is very susceptible to fat, the phosphorus may be given in glutoid capsules, of which each 0.5) contains 1 % phosphorus oil and is dissolved in the intestines. According to the above authors, small and long-repeated doses are preferable, as the phosphorus acts only when every gastric disturbance is avoided. Our results with this treatment are too meagre and not positive enough to permit of any conclusions. They have, however, encouraged us to continue our experiments on a greater plan.

CONTRIBUTIONS TO THE KNOWLEDGE OF INTRACRANIAL COMPLICATIONS OF EAR DISEASE.

By Dr. GEO. LEHR, Clinical Assistant.

Translated by Dr. Richard Jordan, New York.

NOTWITHSTANDING the gratifying results which the surgical treatment of intracranial complications of ear diseases has obtained of late, we cannot but confess that our diagnostic ability and operative procedures are still in need and capable of further improvement. From this point of view it seems desirable to record all such cases in detail. At the same time such complete and continuous publications from the different aural clinics will give us a better insight into the relative frequency of cerebral abscess, sinus-thrombosis, meningitis, and their combinations.

The following report comprises all cases of intracranial otogenous suppuration treated in the Rostock Aural Hospital since Professor Koerner took charge of it, November, 1896. These publications will be continued from time to time.

All cases not previously reported will be given in full with the following two restrictions:

1. The incurable cases of diffuse leptomeningitis will be but briefly mentioned unless they show features of particular interest that warrant a full report.

2. Of the numerous cases of pachymeningitis externa we shall include only extradural abscess, *i. e.*, the formation of

[1] From the Ear and Throat Clinic of the University of Rostock.

pus and granulations between the bone and dura mater or sinus.

Wherever the bone disease merely reaches the dura or sinus, even where they are discolored and granulating, but without the formation of pus or causing symptoms of sinus-phlebitis or pyæmia, neither diagnostic nor therapeutic considerations would justify the report of such cases, as they furnish no particular symptoms and require no other treatment than the removal of the diseased bone.

We wish to emphasize this distinction as we find that quite a number of cases of simple pachymeningitis externa have been reported as extradural abscess — an error which might easily confuse our views on the frequency of extradural abscess.

Of the intracranial suppurations observed in the period referred to, the following have been previously reported:

1. *Phlebitis of sinus petrosus sup. and meningitis. Death.* (Koerner, *Die otit. Erkrankungen*, etc., 2d edit., p. 71.)
2. *Pyæmia after acute mastoiditis from measles. Death.* (Koerner, THESE ARCHIVES, vol. xxvi., p. 294.)
3. *Phlebitis of sinus transversus from cholesteatoma of petrous bone. Recovery.* (Preysing, *ibid.*, xxvii., p. 404.)
4. *Sinus-phlebitis from cholesteatoma. Operation. Meningit. serosa ventricularis ac. Death.* (*Ibid.*)
5. *Sinus-phlebitis with pulmonary metastases. Leptomeningitis. No operation. Death.*
6. *Phlebitis of sinus transversus. Sepsis. Operation. Death.*
7. *Sinus-phlebitis and pulsating perisinuous abscess from cholesteatoma.* (*Ibid.*)
8. *Phlebitis of sinus cavernosus in chronic mastoiditis. Recovery after mastoid operation.* (*Ibid.*)
9. *Extradural abscess in acute mastoiditis. Operation. Recovery.* (*Ibid.*)
10. *Acute extradural abscess. Operation. Recovery.* (*Ibid.*)

Of the cases of **leptomeningitis purulenta** which have not yet been published, the following will be briefly mentioned:

11. Fritz H., aged forty-one, comes to clinic January 31, 1896, with pain in both ears after influenza.

Treatment. Double paracentesis and evacuation of pus. Pat. is lost sight of until March 5, '96, when he appears again with double mastoiditis and cerebral symptoms.

Operation, which reveals an enormous destruction in both mastoid processes, does not stop the progress of the meningeal infection. Death, March 9, '96.

Post-mortem: Extensive suppurative infiltration in spongiosa around hiatus of can. Fallop. On account of this peculiar propagation in the spongy bone beneath the corticalis, this case together with a similar one (No. 19) will be reported in full in a separate publication.

12. Marie N., aged thirty-eight. Rec. Feb. 26, '98. Otorrhœa duplex since early childhood. Mastoid operation. Both mastoids represent mere shells filled with pus and granulations. Death March 26, '98, from meningitis.

Autopsy: Basilar meningitis, probably started from a small deep-seated extradural abscess in the posterior fossa.

13. Emma L., aged twelve. Otorrhœa for two years from diphtheria. Acute exacerbation. Family physician opened periosteal abscess on left mastoid. Chill. Sent to hospital Aug. 26, '98. Mastoid operation: Mastoid, antrum, and attic full of granulations, which are removed. Death on third day after operation with meningitis. Transition of inflammation into cranium through carious tegmen antri et tympani.

The following case of leptomeningitis deserves a more detailed report:

14. Shotgun injury of temporal bone followed two years later by middle-ear suppuration. Operation: removal of ball. Second operation ten months later for mastoiditis. Meningitis. Death.

Gustave D., aged thirteen. Shot himself accidentally in left cheek, Dec. 31, '94 (Flobert rifle, cal. 6 *mm*). Considerable hemorrhage from wound and ear canal. No other symptoms noticed. Wound healed promptly within two weeks. In the fall of '96 the left ear begins to discharge. No pain, but tinnitus and deafness. Pat. asks clinical advice April 15, '97. Muco-purulent discharge in left meatus. Ball visible in upper osseous wall imbedded in granulations, firmly adherent. Operation the same day: Ball chiselled out after temporary displacement of auricle. Tym-

panum found full of granulations enveloping the dislocated hammer; incus not found. May 9, '97, dismissed from hospital; still slight discharge and granulations in posterior part of tympanum.

Pat. paid irregular and infrequent visits to clinic until April, '98, when he returned complaining of great weakness, frequent chills, and dizziness.

Rec. April 29, '98. Status: Haggard appearance, slight vertigo. Profuse discharge from left ear. Canal stenosed through bulging of posterior wall. Swelling and fluctuation above and behind auricle. Temp. 37.5°—C.

Apr. 30th.—Mastoid operation. Subperiosteal abscess. Carious fistula leading to external canal. Large mastoid cells and antrum filled with pus and granulations. Incus found in antrum, small piece of metal in upper meatus wall near recessus epitympanicus. Wound left open. Plastic operation deferred.

May 1, '98.—Temp. in early morning 36.8° C., rises rapidly to 39.6° C. Lumbar pain. Wound of normal appearance.

May 2d.—Temp. mrg., 40.4°, P. 150; T. evg., 39.2°, P. 105. More backache. Restless.

May 3d–6th.—Temp. varying from 37.7–39.0° in the morning to 39.6–40° at night. Pulse about 100 p. min., regular. Condition stationary. No paralysis, no diplopia.

May 7th.—T. 38.0°, P. 100. Pain in neck, but no rigidity. Lumbar puncture in two places negative (no fluid obtained); diarrhœa. T. at night 40.1° C.

May 8th.—Temp. 37.4–40.1° C. P. 95–140.

May 9th.—Temp. 40.1–37.5° C. Fundus of eye normal.

May 10th, 4 A.M.—Temp. 37.4.° Pulse 140 and more, very small and feeble. Restlessness, slight rigidity of neck. Headache.

8 A.M. Temp. 39.4°, P. 120. At noon slight delirium. Sudden convulsions and spasm of respiratory muscles. Pulse 155 p. min., felt and counted two to three minutes after respiration stops. Artificial respiration without avail. Death.

Post-mortem: Extensive basilar meningitis, especially about chiasma. Dura on petrous bone appear intact and so do the facial and acoustic nerves. Removal of petrous bone not allowed. Way of suppuration from middle ear to cranium not found.

All other organs are healthy.

Remarks: With the ball fixed in the upper wall of the meatus outside the middle ear and the suppuration having

followed the injury after nearly two years, there did not seem to be any indication to lay bare all the cavities of the middle ear after the foreign body was removed. If the patient had not been lost sight of, the slight remaining discharge would probably have been cured or at least the ensuing mastoiditis been recognized and operated for in time. The meningitis showed the symptoms of septic infection and could not be safely diagnosed before the terminal respiratory convulsions.

Very gratifying are the surgical results in the following cases:

15. Cerebral abscess in right temporal lobe. Operation. Recovery.

Sophie S., aged twenty-three, servant; since her first year the right ear has been discharging continually. On December 5, 1898, she took sick with severe frontal headache, vomiting, drowsiness, slow pulse; discharge of ear increased and sanguinolent. Received December 14, 1898. Status: face very pale. Questions answered slowly and with hesitation. No real drowsiness. Pulse 60 p. min., small, regular. Temperature 37.1° C. Tongue coated, fœtor ex ore. No earache but headache on right side near vertex.

Right tympanum full of granulations. Pus not offensive. No paralysis. No hemiopia. Pupils equally wide, reaction sluggish, beginning neuritis optica.

Operation (same day): Middle ear full of granulations and cheesy purulent material; hammer carious. Tegmen antri partially destroyed; dura covered with granulations. Dura is laid bare in the whole granulating area; it appears dull, discolored, is tense but shows pulsation. Incision of dura and exploration of temporal lobe with knife in three different directions to a depth of 3 *cm.* No pulse found. Brain substance pale, only slightly bulging into the opening. Cross-incision horizontally backward. Sinus exposed, appears healthy. Attempt to lay bare cerebellum is given up on account of severe hemorrhage from the injured emissary vein.

Wound packed with iodoform gauze.

December 15th–18th.—General condition fair. No more vomiting. Reaction to external impressions is sluggish. Temperature 37.0–37.6° C. Pulse 90–100.

December 19th.—Great apathy. No paresis, no rigidity of neck. Temperature 38.0° C. Pulse 70.

December 20th.—Last night very restless, moaning; increased somnolence. Pupils wide, without reaction. Temperature 36.2° C. Pulse 64.

Second attempt to find the abscess, now supposed to be cerebellar because of the absence of crossed hemiplegia and in view of the failure of the previous exploration.

Cerebellum laid bare in sinus angle. Probing into cerebellar substance with scalpel in different directions with no result. Temporal lobe is again inspected and found pulsating. Several renewed probings are unsuccessful until at last an incision straight inward strikes the abscess at a depth of 4 *cm.* Incision of dura is enlarged and the finger introduced into the abscess, which is tortuous, the size of a hen's egg, and apparently without membrane. The purulent matter is offensive, thin, intermixed with numerous creamy flakes—quantity about two tablespoonfuls. Cavity drained with iodoform gauze. Immediately after the operation the pupils are narrow and responsive, the pulse is fuller and more frequent, 98 p. min.

December 21st.—Patient feels very buoyant, laughs, and asks for food. Pulse varying from 76–100, but strong. Temperature normal.

December 22d.—Dressing changed. About one teaspoonful of pus drains out after removal of gauze.

Eye-fundus: Disc hyperæmic on both sides; the nasal edges are blurred.

December 23d.—Rubber drainage tube inserted.

December 25th.—All well.

December 26th.—At night slightly restless, in the morning drowsy. No objective symptoms.

Two drainage tubes put into abscess cavity, one into anterior, the other into posterior part.

December 27th.—At night much moaning, vomiting; very restless. This morning: somnolence, slight rigidity of neck. No paralysis. Temperature 38.6.° Pulse 100.

December 28th.—Still drowsy, but no more vomiting or rigidity of neck. Strength of left arm seems diminished.

December 29th.—Small prolapse of brain. Anterior drainage tube left out, posterior one shortened. In changing the latter some pus drains out.

December 30th.—Much better. Intellect clear. Weakness of left arm has disappeared.

Thereafter undisturbed recovery.

January 10, 1899.—Prolapse much smaller. After-treatment and daily dressing through ear canal.

February 22d.—Wound above and behind ear firmly cicatrized. Middle ear epidermized.

February 28th.—Dismissed as cured.

March 21st, April 6th, July 12th.—Patient and ear in excellent condition.

Remarks: The symptoms were those of increased intracranial pressure caused by a localized process. The rapidity of their appearance, the absence of a distinct choked disc, and the presence of a suspicious disease of the corresponding middle ear tended to exclude an intracranial tumor and pointed strongly to the diagnosis: cerebral abscess. As localized cerebral symptoms, especially crossed hemiplegia, were missing, and as the dura of the temporal lobe showed distinct pulsation, we were inclined to suspect the abscess in the cerebellum. Nevertheless it was found in the temporal lobe. Not without interest are the signs of cerebral irritation which occurred a week after the evacuation of the abscess, caused undoubtedly by the pressure of the drainage tubes. After the removal of the latter they disappeared promptly.

16. Enormous extradural abscess in posterior and middle cranial fossæ. Total destruction of transverse sinus and extensive destruction of dura; deep intradural abscess between second and third temporal convolutions. Operation. Recovery.

Anna L., aged thirty-six, had otorrhœa sin. since childhood, after measles. In the fall of 1895, acute exacerbation with profuse discharge.

October, 1898.—Influenza. November 10th: Severe earache, fœtid discharge, dizziness.

Rec., *Nov.* 26, 1898.—Ear-bandage soaked with very offensive pus. Continuous flow from ear canal of a thin, sanguinolent matter intermixed with gas bubbles. Mastoid tender on pressure. Gait staggering; intellect sluggish; no paresis. Pulse 76 p. min., small but regular. Temp. 36.8.° Eyes: pupils equally wide, react promptly, horizontal nystagmus; both discs swollen, edges blurred; veins enlarged and tortuous, hemorrhagic spots in both retinæ, more in the right.

Cranial operation: Corticalis sclerotic, ½ *cm* thick. Large cavity beneath is lined with cholesteatomatous membranes and filled with discolored but not fœtid granulations. While opening this cavity a large quantity of very offensive pus with gas bubbles rushes suddenly out from behind. Incision extended horizontally backward. Posterior part of squama temporalis and post.-inferior angle of os parietale removed. Lamina vitrea appears rough and is partly detached from diploë. The dura beneath is covered with granulations. Occipital lobe and upper cerebellum are separated by a deep horizontal furrow caused by the complete destruction of the outer sinus wall. Near the sinus-knee a necrotic piece of sinus is found, 1½ *cm* long, and comprising nearly the whole circumference (its anatomical identity is confirmed by microscopical examination). Dura of temporal lobe is partially destroyed; the brain convolutions are clearly visible, pia mater is covered with granulations. Pus is oozing out from between the two lower temporal convolutions, and after separating them a small intradural abscess is found containing half a teaspoonful of pus. Radical operation of middle ear is postponed. Wound packed loosely with iodoform gauze. For two days after the operation considerable discharge of liquor cerebro-spinalis, which demands frequent renewal of outer dressing.

November 29th.—Patient in good condition; no headache, no vomiting. Pulse 96, regular; temp. 36.0°. Dressing changed on operating table. Whole scalp very œdematous except a small area around the right (healthy) ear.

Discharge of pus so copious that counter-incision is made near prominentia occip. From the grayish-white bone oozes discolored blood. The diploë is congested and discolored, tabula vitrea partly destroyed, partly detached from diploë. The boundary line of abscess and granulations is reached near the torcular. The removal of all the rotten bone results in an enormous defect in the skull, extending from the mastoid up to 1.5 *cm* from the torcular. It is 6 *cm* wide posteriorly and 4 *cm* wide farther in front. The abscess was bordered all around by an uninterrupted wall of granulations and the disintegration of the bone was confined to the same limits. Of the whole lateral sinus no trace could be found. After the operation a severe œdema of the right orbital region developed, which disappeared after forty-eight hours.

General condition excellent. Wound is dressed every second

day and heals rapidly. Neuritis optica subsiding; January 14, 1899, fundus nearly normal.

January 16th, 1899.—Wound healed, except small fistula on mastoid leading into the cholesteatomatous cavity. Radical operation: Cholesteatoma, which lines the whole mastoid from the tip to the antrum, is thoroughly removed and the cavity curetted, enlarged, and polished in the typical way. Plastic: large flap is formed of membranous canal (Stacke) and tamponed against the roof of the cavity. From the external part of the membranous canal and the cymba conchæ a smaller flap is formed which is turned backward and sewed against the cut surface of the auricle.

February 20th.—Retroauricular opening and middle ear dry and epidermized.

February 23d.—Discharge from fistula near the torcular. Incision leads to a deep recess, in which a strip of gauze is found and removed.

February 28th.—Wound completely healed. To cover the opening in the skull a cap made of Stent's mass is fitted to it.

March 6th.—Patient dismissed as cured.

Last seen July 13th, in excellent condition.

Remarks: It could not be doubted that this case represented an intracranial suppuration, but whether we had to deal with a cerebral or a large extradural abscess was well-nigh impossible to decide. The operation unveiled a suppurative process — extensive and complicated beyond expectation. Besides the enormous destruction of sinus and dura the granulations on the pia mater and the abscess between the cerebral convolutions are of particular interest. Our knowledge of such intradural abscesses is confined to a small number of cases (Ceci, Barker, MacEwen).

As a rule, the inflammation spreads quickly through the meningeal meshes and the formation of a wall of granulations on the pia mater is of rare occurrence. Remarkable is the extensive destruction of the parietal bone apparently caused by the long-continued influence of the extradural abscess.

Noticeable for their absence were symptoms of local pressure, as hemiopia, crossed hemiplegia, and aphasia, which have been observed in similar cases.

The healing of the enormous wound took place in a comparatively short time, resulting, as was to be expected, in a large defect of the cranium; the latter was sufficiently protected by a simple prothesis of Stent's mass.

17. Sinus-phlebitis in acute necrosis of mastoid and temporal squama after scarlet fever. Operation with ligation of jugular vein. Recovery.

Child, E. B., eight years old, developed scarlet fever three weeks ago. After two weeks, both ears became affected. The family physician made a Wilde's incision for left mastoiditis and sent the child to the clinic.

Received September 20, 1898; looks very ill. Eyelids and ankles œdematous; skin red, desquamating. Temperature 37.8° C. Urine contains albumen, epithelium cells, and leucocytes.

Both ear canals full of pus; behind left auricle, an incision of 1 *cm* in length discharging greenish pus, mastoid tender on pressure. Immediate mastoid operation. Corticalis discolored, pale. No fistula; cells full of offensive greenish pus. Granulations in antrum, which is curetted; mastoid tip removed.

Dura and sinus laid bare. The latter appears congested and thickened; there is a small discolored spot near its upper knee. Iodoform gauze. Temperature, before operation, 36.0°, rose two hours later to 41.8°, went down to 36.4° during the next twelve hours, and then rose again rapidly to 39.8°. The chart continued to show this intermittent type during the following days. There were no chills or profuse perspiration.

Second operation September 23d: Sinus laid bare more extensively; emissary vein is torn from it accidentally. Through the gap a solid thrombus is visible in the sinus; a few drops of pus ooze out. In the attempt to remove the outer sinus wall a severe hemorrhage occurs apparently from behind, which demands immediate tamponing. A series of enlarged glands alongside the sterno-cleido muscle are removed and the jugular vein exposed. It is empty and collapsed. At the lowest point it is cut between two ligatures. Wound of neck is sewed up.

On account of the continued intermittent fever, which rages from 35.9–41.2° C., an attempt is made on September 27th to change the dressing; it causes a renewed hemorrhage from the sinus. Ligation wound on neck healed by first intention.

Intensity of fever subsides gradually, varying during the following two weeks from 37.0–38.0° C.

October 4th.—First change of dressing. Healthy granulations everywhere. Hereafter daily dressing in the usual way.

October 13th.—Patient complains about pain behind right ear, which had been discharging quite freely all the time. Mastoid is tender and slightly swollen. At the same time a swelling appeared above the left ear, extending over the whole mastoid muscle. No fluctuation. Temperature, 398. ° C.

October 14th.—Operation. Left ear: Incision through the infiltrated parts. A necrotic piece of bone is found above the linea temporalis, covered with sluggish granulations. After its removal, the dura is exposed and appears normal. No pulsation can be felt. Curettement. Iodoform gauze. On the right side a Schwartze operation is performed. The whole mastoid from the tip to the antrum and the dura is found very pliable, its cells partly destroyed and filled with granulations. Dura looks very red and congested. After-treatment in the usual way; healing progresses favorably.

December 7th.—Both mastoid wounds closed.

February 1st.—A small superficial abscess on the left mastoid requires incision and curetting.

March 29th.—Child dismissed from the hospital in the best of health.

Remarks: Remarkable is the very early development of sinus-phlebitis in acute mastoiditis. The necrosis of the temporal squama is not infrequently seen in infants, but its occurrence in older children is rather rare. The glandular swelling on the neck alongside the jugular vein was a symptom of the primary disease (scarlatina) and not caused by sinus-phlebitis; for the exposed jugular vein showed no symptoms whatever of inflammation. More difficult would it be to explain the continuance of the fever after the ligation of jugular vein. It could hardly be that infective matter from the sinus was carried into the system. Perhaps the nephritis was responsible for it. The glandular swelling also and the mastoiditis of the other side have to be taken into consideration.

18. Sinus-phlebitis in acute mastoiditis. Operation. Recovery.

Mr. K., aged twenty-eight, received March 24, 1898.

March 16, 1898.—Pain in left ear, discharge three days later; pain behind the ear followed, extending over the whole side of

head, especially at night. He alleges to have been unconscious once and to have had several chills. Status: Left meatus filled with muco-purulent matter, which pulsates out of a perforation in anterior-inferior quadrant of Mt. Upper-posterior part of membrane bulging, is incised. Soft parts over mastoid are infiltrated and very tender on pressure, particularly toward foram. mast. Temp. 39.0° C. Pulse 80 p. min.

March 25, '98. Operation: Corticalis discolored, congested. After the first stroke of the chisel, pus pulsates out. Bone beneath corticalis friable. Large cells are filled with granulations; but little pus. Antrum large, full of pus and granulations which are curetted. The bone disease extends to the knee of the sinus. Here pus wells out from between the bone and the sinus; sinus wall is partly destroyed, showing a disintegrated thrombus inside. The latter as far as it appears decayed is scraped out in both directions. Sinus wound and antrum are packed separately. Temp. after operation 37.1°, rises to 39.0° in afternoon.

March 26th.—Temp. 37.7–38.3°. No headache. Uninterrupted recovery.

April 4th.—Wound and membrana tympani healed up.

Remarks.—Here again the rapid development of a sinus-thrombosis in an acute mastoiditis is of notable interest.

19. Extradural (perisinuous) abscess in acute mastoiditis after typhoid fever. Operation. Recovery.

On account of the singularity of the primary bone disease, this case, together with the somewhat similar one No. 2, will be reported elsewhere.

It does not seem proper to add general remarks to this series of only nineteen cases, but it might be well to point out the comparative frequency of severe intracranial complications in acute and recent suppurations of the middle ear and petrous bone, and it might further be stated that of the nineteen cases all but three had intracranial complications before they came under our treatment. Two of these, Nos. 11 and 14, had been in our care previous to the intracranial infection, but had stayed away from the clinic until after the development of cerebral symptoms.

Only in one case (No. 12) it is possible, but not certainly proved, that the turn for the worse took place under our

treatment shortly after the operation for the primary disease of the temporal bone.

Of the nineteen cases, three were received with so severe pyæmia or sepsis that the operation seemed almost hopeless. One of them (No. 5) died before anything could be done, and two died immediately after the operation performed as a last resort to save them. Five cases succumbed to an inoperable diffuse leptomeningitis purulenta; one (No. 4) died from meningitis serosa ventricularis after an operation for sinus-phlebitis.

In the remaining ten cases, the intracranial suppuration was cured. These recoveries comprise one cerebral abscess (temporal), one intra- and extradural abscess with destruction of the transverse sinus, four phlebo-thromboses of the sinus transversus, and one phlebitis of the sinus cavernosus. The latter case got well after an operation for the primary mastoiditis without an intracranial operation.

The writer is indebted to Prof. Körner for inviting him to prepare the above paper as well as for assisting him in doing so.

EXAMINATION OF THE PUPILS OF THE MUNICIPAL DEAF-MUTE SCHOOL AT DANZIG, GERMANY.

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Translated by David Greene
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THE results of my examinations on the occasion of the introduction of aural teaching in our municipal deaf-mute school are hereby made public in the hope that this contribution, although it is but small, may promote the cause of deaf-mute teaching. So far, but few examinations of the ears of deaf-mutes by means of the continuous tone-series have been reported. Bezold-Edelmann's continuous tone-series enables us to convey to the ear every tone that it is capable of perceiving, and to define the limit of the capacity of any ear. My opportunities for literary research in this provincial town being limited, I chose Bezold's masterly examinations of deaf-mutes for my guide, and desire that my efforts in this regard be considered supplemental to his. I extended my investigations by using both Bezold's continuous tone-series and Urbantschitsch's harmonica for the purpose of comparing the two scales with each other.

In our school, which furnishes the material for my examinations, there were, during the winter term of 1898–99, thirty pupils—sixteen being boys and fourteen girls. All these pupils were examined, but the following schedule comprises the results of the examination of twenty-nine pupils or fifty-eight ears only, because in the case of one girl the hearing was but slightly impaired, as the result of chronic bilateral suppuration of the middle ear.

In the following tables the pupils are designated in the

order in which they were examined, by the numbers 1 to 29, and the ears by r (right) and l (left).

The investigation was commenced by taking down the history of each case, and the hereditary and consanguineous conditions as stated by one of the relatives, in most cases by the mother. Next followed otoscopic examinations of the adjacent organs, tubes, cavities of the nose, and pharynx. The otoscopic examinations preceded the functional tests for the purpose of ascertaining the presence of obstructions to sound-conduction—cerumen, foreign bodies, etc.—and removing them. Next followed the functional examination with the continuous tone-series and Urbantschitsch's harmonica, which I had perfected by extending the scale in both directions, so that it comprises the full notes from C_1 to f^5. I proceeded with more than ordinary precaution, which I considered advisable in the case of deaf-mutes to avoid errors. After instructing the pupil to raise his hand every time that he feels a sensation of hearing when the tone-producing instrument is brought near his ear, he was placed before the examiner with his face turned away and his eyes covered with a broad bandage, so as to prevent him effectually from seeing what is going on. The hair was brushed back from the ear which was to be examined, and any hair that would not stay back was cut off, so as to avoid any possible contact with the tone-producing instrument. The other ear was closed tightly with the end of the finger of an assistant, which finger had first been dipped into liquid paraffin. Care was also taken not to bring the instrument to the ear rapidly and suddenly, so as to avoid any movement of air which might be mistaken for sensation of hearing. Deaf-mutes are anxious to hear and always ready to believe that they do hear. Their sense of touch is very acute, as is evidenced by the following incident. One of the boys raised his hand regularly every time that the tuning-fork was brought near his ear, whether it was vibrating or not. The bandage over his eyes was carefully examined and properly adjusted, the hair was brushed back of the ear, the tuning-fork was brought near his ear slowly and carefully to avoid any motion of the air, and in spite of all, the

boy would raise his hand when the tuning-fork was not vibrating. The mystery was solved at last when I warmed the tuning-fork in my hand. The cold metal had caused a sensation in the boy's ear, which he mistook for hearing.

The examination proceeded in this careful way, generally beginning with the right ear, and using the deepest tone of tuning-fork 6 of the continuous tone-series dis, and thence ascending and descending in the scale. Every tone in the continuous tone-series that was heard was marked red in the schedule, the same as Bezold indicated it in his examinations of deaf-mutes, whereas every perceived tone of the harmonica was marked blue. Those sections of the range of hearing which were perceived only when the tuning-forks were struck hard, or when the pipes were blown hard, were designated by broken lines. Thus each organ of hearing received its own schedule, which Bezold justly considers an advantage.

After completing the examination by means of the two scales, I tested the acuteness of hearing in each octave with c and g of the unweighted tuning-forks of the continuous tone-series. The result of this test was put down in two decimals for each tone.

Following the example of Bezold, I finally tested each ear with a bell, the tone of which lay between d^4 and dis^4, and recorded the results at the end of the schedule.

The examination was concluded with a test of the ability to hear the speaking voice, which was made during special meetings to avoid fatiguing the pupils. In these tests I was assisted by the principal of the institution, Herr Ravan.

The foregoing description shows that my examinations required a good deal of time, averaging from two to two and a half hours for each pupil. All the pupils were submitted to a second test, and to my gratification I found that the results agreed with those of the original examination.

Among the twenty-nine deaf-mutes who were examined, there were, according to the statements of their relatives:

11 congenitally deaf, viz.: Nos. 9, 12, 16, 19, 20, 23, 24, 25, 26, 28, 29—or 37.9%

15 had acquired deafness, viz.: Nos. 1, 3, 4, 5, 6, 7, 8, 10, 13, 14, 15, 17, 18, 22, 27—or 51.7 %.

3 were doubtful cases, viz.: Nos. 2, 11, 21—or 10.4 %.

The causes of deafness were:

In 4 cases, Nos. 7, 13, 18, 22, or 26.7 %, cerebro-spinal meningitis.

In 3 cases, Nos. 6, 8, 10, or 20 %, inflammation of the brain.

In 3 cases, Nos. 1, 14, 15, or 20 %, convulsions.

In 2 cases, Nos. 3, 17, or 13.3 %, scarlatina.

In 1 case, No. 27, or 6.7 %, chicken-pox.

In 1 case, No. 4, or 6.7 %, eruption of the scalp.

In 1 case, No. 5, or 6.7 %, unknown.

The figures for acquired deafness, 6.7 % from unknown causes, agree with those of other investigators; thus Wilhelmi found 6.3 %, Schmaltz 7.1 %, Barth 6.6 %, Bezold 5.8 %; Falk only, with 27.8 %, shows different results.

In three cases, Nos. 2, 11, 21, or 10.4 %, the reports of the relatives were incomplete, and it had to be left undecided whether deafness was congenital or acquired. In this regard also the statements of different investigators do not agree: Wilhelmi found .8 %, Falk 2.8 %, Hartman 3.9 %, Lemke 6.9 %, Bezold 7.6 %, Frankenberger 12.5 %, Schmaltz 13.2 %, Roller 15.1 %, and Mygind 21.5 %. My figure, 10.4 %, differs by 1 % only from the average of the foregoing percentages, which is 9.4 %.

Adenoid growths were found during my examination as follows:

2,	Nos.	26, 27,	in children,	7	years of age
2,	"	20, 25,	"	8	" "
2,	"	6, 15,	"	9	" "
1,	No.	17,	"	10	" "
1,	"	14,	"	11	" "
1,	"	9,	"	14	" "
1,	"	1,	"	15	" "
1,	"	28,	"	16	" "

Depression of the drum-head was found in eighteen ears, bilateral in each case, therefore in nine children or 31 %. The depression of the drum-head occurring on both sides

proves that the cause was the same on both sides, viz., adenoid growths.

Ten ears, or 17.2 %, showed traces of former deafness on the drum-heads—namely, scars in 3 ears, or 5.2 %—Nos. 15 r, 21 r, 26 l; opacity of the drum-head in 7 ears, or 12 %—3 r, 3 l, 4 r, 4 l, 12 r, 12 l, 24 r.

In two ears, or 3.4 %, chronic suppuration of the middle ear was found, case 24 l showing perforation of the drum-head, and case 15 l polypus with perforation.

The examination of the nasal passages revealed suppuration in seven instances, or 12 %—bilateral in 9, 22, 28; unilateral in 4 l.

The tests with the continuous tone-series showed 10 cases of total deafness, being 17.2 %, and 48 cases of partial deafness, or 82.8 %.

These results are more favorable than those of Bezold, who found 30.4 % of total and 68.4 % of partial deafness.

The cases of partial hearing are divided into five groups only. Bezold gives six groups. Group III. of his scale—defective regarding the upper part of the scale—is omitted, no cases of that kind having been found in my examinations.

Group I. Small areas to the extent of 2½ octaves shows	3	ears, or	5.2 %
Group II. Intermissions	13	"	22.4 %
Group IV. Defects at the upper and lower ends of the scale	12	"	20.7 %
Group V. Defects at the lower end of the scale extending over 4 octaves	2	"	3.4 %
Group VI. Defects at the lower end of the scale, under 4 octaves	18	"	31.1 %
	48	ears, or	82.8 %

Taking them individually,

Bilateral deafness was found in	3	pupils, or	10.3 %
Unilateral deafness in	4	" "	13.8 %
Partial hearing in both ears in	22	" "	75.9 %
" in one ear	4	" "	13.8 %

Bilateral total deafness was found in 3 cases only—7, 11, 20—or 10.3 %; partial hearing in one or both ears in 26 cases,

or 89.7 %. These figures approximate those of Bezold, who found

15 totally deaf, or 19 % . . .
63 with partial hearing, or 79.7 %

Special interest attaches to the question of the relation between the degree of partial hearing in the different groups and the different forms of deafmutism.

In table I. the degrees of partial hearing are arranged according to the forms of deafmutism.

TABLE I.

Group.	Number of Ears.	Congenital Deafness.		Acquired Deafness.		Doubtful.	
		Number.	%	Number.	%	Number.	%
Total Deafness...	10	3	30	5	50	2	20
I.....	3	1	33.3	2	66.7	—	—
II.....	13	6	46.2	7	53.8	—	—
IV.....	12	5	41.8	7	58.2	—	—
V.....	2	—	—	1	50	1	50
VI.....	18	7	38.9	8	44.4	3	16.7

It is to be seen from this table that total deafness is to be found more frequently among those who acquired deafmutism than among those who were born with it. That the reverse is the case in regard to partial hearing is not at once apparent, yet comparing the totals we find for congenital deafmutism 145 octaves for 22 ears, against 116½ octaves for 30 ears in cases of adventitious deafness, or an average of 6.6 octaves for each ear of the congenitally deaf and 3.9 for those whose deafness has been acquired. From this it seems probable that the doubtful cases are mostly congenital deaf-mutes.

The presence of analogous defects in both ears furnishes interesting points regarding the extent of disease-processes.

In 10 totally deaf ears I found.....						6	bilateral, or	60 %
In Group I. I found among			3	ears.....		0	"	—
"	II.	"	"	13	"	.....	6	"	46.2 %
"	IV.	"	"	12	"	.....	6	"	50 %
"	V.	"	"	2	"	.....	0	"	—
"	VI.	"	"	18	"	.....	12	"	66.7 %

Compare these figures with those of Bezold, who found:

Among 48 totally deaf ears						30	bilateral, or	62.5 %
In Group I. among	28	totally deaf	ears...			10	"	" 35.7 %
" II. "	20	"	" ...			8	"	" 40 %
" III. "	1	"	" ...			—	"	" —
" IV. "	8	"	" ...			4	"	" 50 %
" V. "	18	"	" ...			6	"	" 33.3 %
" VI. "	33	"	" ...			22	"	" 66.7 %

The fact that my figures are almost the same as Bezold's shows the correctness of his conclusion that "the percentage of bilateral analogous defects of hearing has an important bearing on their nosological identity."

The results of the tests with the harmonica are not arranged in groups but are divided similar to Bezold's.

Among 10 ears which appeared totally deaf during the tests with the continuous tone-series, there were 6 (20 r, 13 l, 7 r, 20 l, 7 l, 10 r), or 60%, which showed small remnants of hearing, ranging from ½ to 3 octaves, during the tests with the harmonica. All remnants of hearing are within the small and great octaves. Four ears, or 40%, are totally deaf to both the continuous tone-series and the harmonica.

Comparing the tests with the two instruments respectively, we find that among the 58 ears there were 10 ears, or 17.2%, totally deaf to the tones of the continuous series, but only 4, or 6.9%, to those of the harmonica. The last named figure, viz., 6.9%, does not differ materially from that found by Urbantschitsch, who states that only 3 out of 144 ears, or 2.1%, were totally deaf to the tones of the harmonica.

Of the 3 ears of Group I., one—10 l—showed a defect at the upper end of the scale of the harmonica. One—8 l—at the upper and lower ends; and one—28 r—a blank. The number in this group is too small to justify conclusions.

In Group II. there are among 13 ears:

1 ear—17 r—which perceived all the tones of the harmonica.

4 ears—19 r, 23 r, 15 r, 19 l—or 30.7%, with defect at the upper end of the scale of the harmonica.

6 ears—16 r, 1 l, 22 r, 4 l, 16 l, 25 r—or 46.2%, with defects at the upper and lower ends of the scale.

2 ears—17 l, 13 r—or 15.4 %, with intermissions in the scale.

This group shows the largest number of ears with defects in both the upper and lower parts of the scale of the harmonica.

In Group IV. there are among 12 ears:

1 ear—29 l—which perceived all the tones of the harmonica.

3 ears—22 l, 3 r, 23 l—or 25 %, with defects of less than two octaves at the lower end of the scale.

4 ears—12 l, 9 l, 9 r, 4 r—with defects of 3½ octaves at the upper end.

3 ears—8 r, 6 r, 3 l—or 25 %, with defects at the upper and lower ends.

1 ear—6 l—with intermissions.

This group contains the largest number of cases—33.3 %—with a defect in the upper portion of the scale of the harmonica.

Of the 2 ears of Group V., 1 ear—18 l—perceived all the tones of the harmonica and 1—2 l—showed a slight defect in the lower end of the scale of the harmonica.

Of 18 ears in Group VI.:

15 ears, or 83.3 %, perceived all the tones of the harmonica.

2 ears, or 11.2 %, with defects in the upper portion of the scale of the harmonica.

1 ear, or 5.6 %, with a slight defect in the lower portion of the scale.

A comparison of the two scales shows some interesting details:

I. Of the 10 ears which are totally deaf to the continuous tone-series, 6 show small areas for the harmonica in the smaller and greater octaves, which proves that the tones of the greater and smaller octaves of the harmonica have the largest amplitude.

II. In all the ears of Group I. the upper limit is higher in the continuous tone-series than in the harmonica. This shows that the tones of the upper portion of the scale of the continuous tone-series have a greater amplitude than those of the harmonica.

III. In most cases the lower limit of the range of hearing

lies deeper in the harmonica than in the continuous tone-series. This shows that the tones of the lower portion of the scale of the harmonica have greater amplitude than those of the continuous tone-series.

After the tests with the tone-series, the duration of hearing of each ear was ascertained for the tones of c and g in each octave. The figures which I obtained differ from those given by Bezold. I found the following:

G vibrates	258	seconds.	According to Bezold,		203	seconds.	
c	"	194	"	"	"	169	"
g	"	209	"	—	—	—	—
c_1	"	273	"	—	—	—	—
g_1	"	206	"	"	"	270	"
c_2	"	145	"	"	"	223	"
g_2	"	127	"	"	"	212	"
c_3	"	115	"	"	"	142	"
g_3	"	132	"	"	"	91	"
c_4	"	49	"	—	—	—	—
g_4	"	24	"	"	"	17	"
c_5	"	8	"	—	—	—	—

It was found that the duration of hearing decreases as the range of hearing approaches the upper end of the scale. This corroborates the supposition that in most cases deaf-mutism is caused by lesions in the organ of Corti.

Next followed tests with a bell, the specific tone of which lay between d^4 and dis^4. The distances at which the bell was heard are given in metres. The room in which the examination was held did not admit of distances over 8 metres, therefore I use the mark > 8 *m* for those who could hear at a greater distance.

The ten totally deaf ears and the three ears of Group I. did not hear the bell.

Of the 13 ears of Group II., 8 ears, or 61.5 %, did not hear the bell; in 5 cases, or 38.5 %, the distance varied between .05 *m* and .5 *m*.

Of the 12 ears of Group IV., 1 ear, or 8.3 %, did not hear the bell. With the remaining 11 ears which heard the bell, the distance varied between .01 *m* and 1.2 *m*.

In Group V., the distance ranged from .4 *m* to 3.25 *m*.

Of the 18 ears of Group VI., 9 ears, or 50 %, heard the bell at a distance of > 8 *m*, and with the remaining 9 the distance varied between .15 *m* and 7.75 *m*.

Comparing the distances in the different groups at which the largest number of cases heard the bell, we find that in:

Group I. none heard the bell;
" II. 8 did not hear the bell;
" IV. 6 from .05 *m* and .2 *m*;
" V. " .4 *m* and 3.25 *m*;
" VI. 9 > 8 *m*.

From this it is to be seen that with the increasing limit of audition, the distance also increases, which proves that Bezold's arrangement of the cases into six groups is proper.

I have now reached the last of my tests, namely, that of ascertaining the ability of deaf-mutes to hear speech. It is advisable to make separate tests of the ability to hear consonants, vowels, and words.

Of the 10 totally deaf ears, 4 ears, or 40 %, perceived the sound of *p* correctly, but none of the other consonants was heard.

In Group I., with three cases, *p* was perceived by 2 ears, or 66.7 %.

In Group II., with 13 ears:

p was perceived by 7 ears, or 58.3 %,
r[1] " " " 3 " 25 %.

In Group IV., with 12 ears:

p was perceived by 8 ears, or 72.7 %,
r " " " 4 " 36.4 %.

In Group V., with 2 ears:

p was perceived by 1 ear, or 50 %,
t " " " 1 " 50 %,
r " " " 2 ears, or 100 %.

In Group VI., with 18 ears:

p was perceived by 15 ears, or 83.3 %,
t " " " 14 " 77.8 %,
r " " " 11 " 61.1 %.

[1] It should be borne in mind that the author refers to the rolling *r* in German.—The Translator.

These large percentages do not prove actual auditory perception of the sounds of *p*, *r*, and *t*, as correctly remarked by Bezold, but simply that these sounds cause tactile sensations which may be easily mistaken for hearing, as any one will notice if he sounds these consonants while holding the back of his hand before his mouth.

Similar results were found regarding the consonant *f*. This was perceived by, totally deaf ear, by 1 in Group II., and by 2 in Group VI.

m, *n*, and *l* were perceived by very few ears only, which, as Bezold explains, is accounted for by the fact that the special tone of these nasal consonants lies within the limit of the lower scale of tones that are not perceived by most deaf-mutes.

n was heard by 1 ear in Group II., which could distinguish all the tones of the scale with a slight intermission from cis^2 to fis^2.

In Group VI., *m* was heard in 3 instances, *n* in 2, and *l* in 1.

The consonant *k* was heard in 2 instances in Group VI.

The consonant *s* and the other sibilant sounds were also heard in Group VI. only, namely, in 4 instances.

In testing the ability to hear vowels, the degrees of pitch were used which Helmholtz has fixed for this class of sounds, viz.:

for *u* tone f,
" *o* " b,
" *a* " b^2,
" *i* " d^4.

The totally deaf ears and those of Group I. did not hear any of the vowels.

In Group II. *u* was heard by 1 ear, being the only one in this group which could perceive the whole lower part of the scale; *a* and *o* were heard by 1 ear which had shown only a slight defect in the lower part of the scale.

In Group IV.:

u was heard by 2 ears,
a " " 4 "
i " " 1 ear.

In Group V.:

u was heard in 1 ear,
o " " 1 "
a " " 2 ears,
i " " 1 ear.

In Group VI.:

u was heard in 17 ears,
o " " 15 "
a " " 17 "
i " " 17 "

The results of the vowel tests with deaf-mutes showed that when a vowel was perceived the tone corresponding to that vowel was also perceived, which proves the correctness of Helmholtz's arrangement of the vowels.

The results of the tests of the ability to hear words were as follows:

Numbers were not perceived by the totally deaf nor by those of Groups I. and IV.—according to Bezold's statement only the numbers 1–10 and 100 were tried. In Group II., 8 and 100 were heard by 15 r. In Group V., 18 l heard all the numbers except 4.

In Group VI.:

5 r heard all numbers.
5 l " " except 2.
18 r " 3, 9, 10, 100.
15 l " 2, 3, 4, 6, 8, 9, 100.

Now, what are the practical conclusions that may be drawn from the foregoing? The tests which have been made in the public schools by Richard, Weil, Bezold, Schmiegelow, and Ohleman have demonstrated that in a considerable number of school children the ears were in a sufficiently diseased state to require the treatment of an aurist. There is, therefore, urgent need of the services of inspecting physicians who are skilled in the examination of the eye and the ear. In the case of deaf-mute children this need is still greater, because the ears of all of them are defective and in many of them the disease which caused deaf-mutism is still active. I found adenoid growths in eleven

deaf-mute children. Such growths hinder correct articulation. Every pupil who articulates badly should therefore have his nose and throat examined.

Chronic suppuration of the nose and of the middle ear claims special attention—all that needs to be mentioned in this connection is that in some instances tubercle bacilli were found in the discharge from the ear. I therefore agree with Bezold that it should be one of the first rules of school hygiene to place children who are suffering from chronic suppuration of the ear under treatment by an aurist. Fortunately the constant warnings of this kind have not remained unheeded, and better attention is now paid to the physical conditions of the pupils of the public schools and of deaf-mute institutions.

Of greater importance is the question which was brought out by the experiments of Urbantschitsch and Bezold, namely, in what way the partial hearing of deaf-mutes can be utilized. It is a well-known fact that a considerable number of deaf-mutes possess sufficient hearing to receive instruction through the ear, and attempts of this sort have been made since Itard and Toynbee. The results of Urbantschitsch in conjunction with the teachers of the deaf-mute school in Döbling, Austria, have excited universal interest, and Urbantschitsch is entitled to great credit. He practised his aural exercises with all pupils, even those who are totally deaf, and insists that hearing can be developed even in apparently totally deaf persons. It remains to be seen how far he is correct. I do not believe that the results will be of much practical value, because all that can be expected is that the deaf-mutes will learn how to make use of the partial hearing that they possess. It may be safely asserted that if portions of Corti's organ have been destroyed by disease, they cannot be restored through aural exercises.

The results of Urbantschitsch's agitation was that the teachers of deaf-mutes introduced his method in their schools before it could be examined by the aurists. No proper selection of suitable pupils for aural exercises having been made, complaints of failure were soon heard on all sides,

and some of the teachers became opposed to aural instruction. Urbantschitsch by his experiments has therefore done very little to advance the cause of deaf-mute teaching.

The interest in this question was increased when Bezold published the results of his examination of deaf-mutes, showing that he had succeeded in defining the limit of hearing of each ear. Thus a guide was furnished for the instruction through the ear. The results obtained at the Central Deaf-Mute Institution in Munich prove that Bezold's method is correct. This method was approved by the State Department of Public Instruction, which decreed that "the semi-deaf and semi-mute receive special instruction with a view of preserving and improving their ability to hear and to speak." Since the instruction through the ear was introduced in the deaf-mute institutions of Bavaria, similar steps have been taken in the schools of other States of the German Empire, and it is to be hoped that, in spite of the opposition from many quarters, aural teaching will soon be carried on in addition to articulation teaching in all the German schools. I repeat again that aural teaching is to form an integral part only of the general system of deaf-mute education without superseding the instruction in and by articulation. Only the semi-deaf and semi-mute are to be taught aurally in separate hours. I hope that the Joint Convention of Aurists and Deaf-Mute Teachers, which is to meet next September at Munich, will solve this question satisfactorily, and that the time will soon come when aurists and deaf-mute teachers will work unitedly for the advancement of the deaf and dumb.

REPORT ON THE TRANSACTIONS OF THE TWENTY-THIRD ANNUAL MEETING OF THE AMERICAN OTOLOGICAL SOCIETY, HELD AT WASHINGTON, D. C., APRIL 13, 1900.[1]

Dr. HERMAN KNAPP, New York, after demonstration of some anatomical specimens, related a case of **extensive acute caries of the mastoid and petrous portions of the temporal bone, on which he operated successfully with restoration of perfect hearing and preservation of the external ear canal and the tympanic cavity.**

He sums up the noteworthy features of the case as follows:

1. In an acute tympano-mastoid suppuration of a healthy man, thirty years of age, who never had had ear trouble before, the tympanum, drum-head, and hearing power were restored, while the destruction went on in the mastoid, and the adjacent third of the petrous portion of the temporal bone, under formation of an outer fistula of the mastoid.

2. Headache and the continuance of the mastoid disease determined the patient to give his consent to an operation which he had formerly refused.

3. The operation, consisting in a total resection of the mastoid, exposing the dura in the posterior cranial fossa, scooping away all the carious bone in the basal portion of the petrous, and carving out with a sharp spoon the bony wall of the facial canal in its whole length through the mastoid, and the entire horizontal semicircular canal, forming a platform from the latter to the frontal semicircular canal, where the caries stopped.

4. The complete and unusually rapid recovery, with integrity

[1] This being the conjoined triennial Congress of the American Physicians and Surgeons, one forenoon only was allotted for the meeting of the Otological Society. The abstracts contained in this report have been kindly furnished by the speakers, for which the editors of these ARCHIVES, in the name of the readers, express their thanks.

of the sound-conducting apparatus, and restoration of perfect hearing. [Operation January 15, 1900; discharged from hospital February 1st; wound closed February 16th; March 1st, H $\frac{6}{24}''$, V $\frac{20}{20}$.]

Discussion.—Dr. DENCH spoke commendingly on the management of Dr. Knapp's case. It showed what excellent results could be obtained if, during the progress of an operation, we modified the general plan in its details according to the conditions we encountered on our way. Clear exposure and competent appreciation of these conditions were the secret of success.

Dr. GRUENING: I would say that in this connection we should not forget that this work has been done by Jansen, of Berlin, who has published a large number of cases of caries of the petrous portion of the temporal bone, and has successfully operated on a large number, so that he certainly opened our eyes to this matter many years ago, and those who know the literature of otology are aware that these operations have been performed by Jansen. I have also performed it in a number of cases knowing that Jansen had precedence in the matter. It is a law of surgery to remove carious bone wherever we meet it. I must say, though, that I have refrained from removing it from around the facial canal. Dr. Knapp, in his case, says he cleaned out everything around the facial canal. After he had found caries in the canal, I think he could probably have opened the bone freely and allowed the extrusion of the carious substance. In these cases we often see that the facial nerve preserves its function. I saw a similar case in Berlin in the clinic of Jansen, where he removed everything, but avoided carefully just that portion of the bone which included the facial nerve. To produce facial paralysis in an operation is a very grave thing, especially in cases of the young, and in female patients.

Dr. RANDALL said there was one point in Dr. Knapp's paper he would like to emphasize, the preservation of hearing. In the serious danger to life which these cases generally entail, the question whether the hearing is saved or not was regarded as of little importance, but the *aurist* who sacrifices the hearing is like the obstetrician who saves only the father. The early as well as the radical intervention in these cases was at times extremely important if we wanted to retain the function of hearing. In the severer cases, the penetration of the carious process to the dura and not infrequently to the petrous portion was the rule rather than the

exception. In at least half of his last one hundred cases he had to lay bare the dura.

As to the facial, he said that with good light and careful cleansing of the field with gauze strips, the facial canal could be well defined and as a rule scraped clean, and even caries removed, without injury to the nerve.

Dr. C. H. BURNETT said that thoroughness in cleaning away all that is diseased was the chief object in operation for chronic ear disease, and Dr. Knapp, having done that in the case he reported, could have obtained healing by first intention if he had desired it. He illustrated his remarks by a case where he obtained healing by first intention after a mastoid operation, but a relapse followed later, as the tympanic cavity had not been thoroughly cleaned out. When this was done by a radical operation, permanent recovery ensued.

Dr. BACON said that he could not second all Dr. Randall mentioned. He had had one or two cases of permanent facial paralysis. He avoided the facial canal wherever possible. In all the cases he had operated on there was considerable hemorrhage and great difficulty in seeing what could be removed with absolute safety.

Dr. KNAPP, replying to Dr. Grüning's remarks, said he was aware that nothing new had been done in the operation he had reported. It was an advanced case of aural disease, but singularly fortunate. The carious destruction of the whole mastoid had extended deeply into the petrous portion, disintegrating the cancellous part of the bone, but not yet affecting the compact osseous structure of the walls of the facial and semicircular canals. The walls of the facial passed, like an untouched ivory rod, from above downward, and as there was no symptom of disease in the facial nerve, no indication presented itself to interfere with the canal. The disintegration stopped at the superior vertical canal, leaving the vestibule and cochlea intact, which accounted for the rapid recovery and the excellent auditory result of the case. Dr. K. said that he also, in his visits to Berlin, had frequently availed himself of the generously given opportunity to witness the superior skill of Dr. Jansen, who in dealing with the extensions of mastoid disease to the petrous portion and through it into the posterior cranial fossa, stood at the head of the pioneers in this field.

Dr. C. H. BURNETT maintains that **chronic ear vertigo** of Ménière's syndrome is chronologically the latest symptom of chronic catarrhal otitis media, being always preceded by profound deaf-

ness and tinnitus. It is due to undue impaction of the stapes in the oval window, as well as to stiffening of the round-window membrane, from the catarrhal condition of the drum cavity. In a normal ear any inward pressure of the stapes upon the labyrinth fluid is compensated by a corresponding outward movement of the membrane of the round window toward the tympanic cavity. Any undue pressure from within the labyrinth by influx of perilymph or endolymph from the cranial cavity is compensated by a corresponding outward movement of the stapes as well as of the round-window membrane towards the drum cavity. All or any of these compensations being interfered with, intralabyrinth pressure is increased, the ampullar nerves are unduly compressed, and reflex phenomena evoked which are termed ear vertigo. As these altered conditions of intralabyrinth pressure are not constant, but vary with the health of the patient and the state of the drum cavity and internal ear, chronic ear vertigo is paroxysmal in nature. As retraction of the chain of ossicles and consequent impaction of the stapes in the oval window, in chronic catarrh of the middle ear, play the greatest part in the production of these vertiginous phenomena by a compromise of the internal ear cavity, Burnett proposes to liberate the stapes from the superposed incus by removal of the latter, through an incision in the upper posterior quadrant of the membrana tympani of the etherized patient. This he has done in 27 cases, giving entire relief from vertigo in every instance.

Dr. RANDALL spoke of the **clinical anatomy of the Eustachian tube,** and the rediscoveries of the Eustachian catheterization as showing need of better appreciation of the known anatomy. Among all the variables of aural topography the position of the tube-mouths may be counted a constant since it is essentially related to bony structures of little varying configuration; and the claims of variation are generally with reference to unrelated nasal and pharyngeal points instead of to the back edge of the hard palate, which is the true landmark. The lumen of the tube is a slit, usually collapsed and at its inner third devoid of the "safety-tube"; while a valve-like fold in its bifurcated lower part serves with the drag of the relaxed palate to insure its closure except in the act of swallowing. Slight variations are to be expected in all points of aural anatomy, but those of the tube having real clinical importance will be very rarely found. Sections, casts, and bone-preparations were used in illustration of the points insisted on.

Dr. HIRAM WOODS, Jr., Baltimore, Md., read the **clinical history of a fatal case of septic sinus thrombosis.** Patient, a boy thirteen years old. Family history of tuberculosis. Measles when he was two years old, followed by right otorrhœa, which has persisted with occasional intervals ever since. Apparently he never has had careful treatment. About the 2d October, '99, after a paroxysm of right earache, had a chill, followed by fever. This was repeated each day till Oct. 5th, when the family physician was summoned, who sent patient to the reporter. On admission the boy was in great pain. T. 101.6°, P. 106. There was diffuse mastoid tenderness, the aural canal was filled with a polyp, while the general appearance of the boy was septic. He had a pyæmic rigor shortly after admission. Save for these constitutional symptoms there were no indications of sinus involvement. Locally the case presented the picture of internal mastoiditis only. Operation was performed next day. Mastoid process was eliminated. The polypus above mentioned sprang from a small area of necrosed bone. The inner wall of the mastoid covering the sinus was soft. Bone was removed, exposing the sinus for a space of an inch and a half. Dura was necrotic, while the external sinus wall was ulcerated, the lumen being plugged above and below by a yellowish, fibrinous clot. This was removed with curette, and good blood currents obtained in each direction. Sinus was closed with plain gauze. On the two succeeding evenings there was an elevation of temp. but no chill. Then, without characteristic change in the thermal line, there developed in the course of ten days a painful swelling in the neck, along the inner border of the sterno-mastoid muscle. A large amount of pus was evacuated from the jugular canal, the vein being found collapsed. After this the T. line became pyæmic. Metastatic abscesses developed in different parts of the body. Death occurred on Nov. 11th. General streptococcus infection was found on autopsy, together with a septic thrombus, closing the clavicular end of the jugular. The paper discussed the general question of ligation of the jugular in cases of septic thrombosis where on operation good blood currents are obtained and there are no symptoms of jugular involvement.

Dr. E. B. DENCH, New York, reported **a case of sinus thrombosis, complicated with cerebellar abscess.**

Discussion.—Dr. GRUENING : I recall at present several cases of thrombosis of the lateral sinus in some of which the thrombus

was removed and in some it was not removed ; in some the jugular vein was ligated and in others not.

The FIRST CASE is that of a soldier returning from Porto Rico, who had typhoid fever, and who, in the course of the fever, developed a thrombosis of the large veins of the leg. He was recovering from his typhoid fever when he was taken with mastoid disease. He had a temperature of 106° and I decided to operate. I found a large mastoid of the pneumatic variety and all the cells were filled with serous fluid, an examination showing that it abounded in streptococci. The lateral sinus was laid bare ; the inner table was still sound, but on it were a large number of these small cells filled with serum. I found that the sinus was absolutely solid. This patient was very weak and I did not think it advisable to proceed any farther. I assumed that it was possible for such a man to have a non-infective clot in his sinus, just as in the veins of the leg. I found to my joy the next morning an almost normal temperature, and he made a rapid recovery with the sinus blocked with this thrombus ; so then there are cases, no doubt, in which the thrombus is non-infective and can be dealt with as in other parts of the body. That is one class of cases.

A SECOND CASE was that of a child who came into the hospital with a history of long-standing otorrhœa. It had had several chills a few days before admission. I found a thrombus of the lateral sinus, not only of the sigmoid portion, but also of the lateral sinus proper, and this extended very far back. It was necessary to expose two and a half inches of the sinus. It was cleaned out completely. The bacteriologist found that the thrombus was non-infective and I concluded that it was not necessary to ligate the jugular vein. The child recovered. So there is a second class of cases where we do actually remove even the non-infected thrombus.

Then a THIRD CASE, that of a young woman, nineteen years old, who came to the hospital with a history of chronic otorrhœa. She had had a great deal of headache, and for the past week before admission several chills. On examination we found caries of the ossicles, caries of the walls of the tympanic cavity ; there was no tenderness ; the bone was thick and I assumed that perforation had occurred into the sinus and that there was probably an abscess. On opening I found a perisinuous abscess. After cleansing it thoroughly I put a needle into the sinus and drew blood, which was found to be non-infective. The patient did not, however, improve. She had still chills and high temperature characteristic

of sinus thrombosis and I then made a large incision into the sinus. There was a free flow of blood from above and below. Nevertheless, I ligated the jugular vein, assuming that there was, perhaps, an incomplete thrombus at the bulb. The patient recovered. So that there is a third class of cases where, though we do not find a clot in the sinus and no evidence of clot on aspiration, still if the temperature continues as the characteristic temperature of infective thrombosis we should still ligate the jugular.

A FOURTH CASE is that of a young woman who came to the hospital four weeks ago with ordinary mastoid disease. I did a complete operation and she did well for the first two weeks, at the end of which time she had very high fever, 104–5°. I put her under ether, examined the sinus, and found it was perfectly healthy. As there was no indication for any operative interference I concluded to wait, assuming that there was pyæmia without thrombosis. This patient then developed a swelling in her knee, had a fluctuating temperature, then she had an inflammation of all the extensor tendons in one hand, and then an inflammation along the tendons in the foot. She died, and I thought that if I had in this case ligated the jugular the result might have been better. So that there is a class of cases where we cannot find the thrombus and yet have all the symptoms of infective thrombosis where it is well to ligate the jugular.

There is no hard-and-fast rule. To say that in every case of this kind we should ligate is not correct, and to say that in no case should we ligate is not correct.

Dr. EDWARD FRIEDENBERG, New York, read a paper on **pneumococcic perisinuitis.**

Discussion.—Dr. GRUENING: In fifty cases of mastoiditis we have had only three cases of pneumococcus infection, and these three cases did well. In the other cases were found streptococcus and staphylococcus. The bacillus of grippe was not found.

Dr. GORHAM BACON, New York, reported a case of **chronic purulent otitis media, followed by an abscess in the temporo-sphenoidal lobe, and also an abscess in the cerebellum; autopsy.**

The patient, Mrs. A. P.——, thirty-two years of age, had suffered at times from a chronic discharge from the right ear, although of late years it had given her no trouble, except that the hearing was defective. For one month prior to her admission to the infirmary she complained of severe pain in this ear, and radiating

pains on the same side of the head. Three days before admission the discharge from the ear reappeared.

For two weeks she has been confined to her bed, and nine days ago she had two chills on the same day. Following the chill there was vomiting, and since that time she has had some nausea and vomiting.

As the pain in her head and ear was severe, her family physician prescribed opiates, and when she came to the hospital, February 13, 1900, she was under the influence of morphine and very stupid. Temperature 100⅘° F. Pulse 80. Respiration 20. Right external auditory canal full of pus, and very little left of the drum-head.

Under ether, that same day, the usual mastoid operation was performed, and pus,—offensive in character,—granulations, and softened bone were removed. No opening could be detected in the tympanic roof, and as it was difficult to make a diagnosis of intracranial complication owing to the administration of the morphine, any further operative interference was postponed.

February 18th.—The pain has continued. To-day she has paralysis of left abducens, paralysis of left side of face, slight left hemiparesis, moderate left hemianæsthesia, left hononymous hemianopsia, and choked discs.

Diagnosis.—Abscess in right temporo-sphenoidal lobe.

Second operation.—Original wound reopened and the incision carried upwards so that the bone could be thoroughly cut away for a considerable area above the ext. meatus. Dura found thickened, but not adherent to the tympanic roof. A small sinus found in the dura with a probe. This was enlarged, and a large abscess found on the right temporo-sphenoidal lobe. About two ounces of pus evacuated.

For several days after the operation the patient seemed to improve, but later the paralysis became worse, the choked discs more marked. Patient very restless, and a diagnosis of probable leptomeningitis was made. The patient lived till March 3d.

Autopsy.—The temporo-sphenoidal lobe presented a large abscess cavity passing well back. It had been well drained. The base of brain presented nothing of special interest. Abscess found in right lobe of cerebellum. Foul-smelling pus and very thick. It appeared to have begun in the dentate body, which it destroyed. It then passed across to the opposite lobe, which it invaded to the extent of half an inch. The ventricles were found normal. No communication could be demonstrated between these two abscess cavities.

REPORT ON THE PROGRESS IN OTOLOGY AND RHINOLOGY DURING THE FOURTH QUARTER OF THE YEAR 1899.

By Dr. A. HARTMANN.

Translated by Dr. Arnold Knapp.

ANATOMY OF THE EAR.

272. Varaglia, S. On the elastic fibres of the drum membrane. *Arch. ital. di Otologia*, vol. ix., p. 49.

272. Elastic fibres are abundant in both the tense and the flaccid portions of the drum, and can be grouped in three kinds: 1. Elastic radial fibres of various thickness; these run in the radiating layers. 2. Elastic circular fibres are most numerous in the periphery and run in the circular layers. 3. Very fine reticular fibres which connect the two varieties. Gradenigo.

PHYSIOLOGY OF THE EAR.

273. Schäfer, K. The determination of the lower limit of hearing. From the psychological seminary of Berlin University. *Zeitschr. f. Psychologie*, etc., 1899, p. 161.

273. According to Schäfer, previous experiments on the absence of overtones in the production of the lowest tones are not convincing. He considers the proof lacking that tones of 16 or perhaps of even lower number of vibrations are audible, though the possibility is not denied. He shows by experiments that even 16 stimulations in a second are capable of producing a tone perception. The lower limit is not exactly determinable, not a sharply defined point, and may show variations with the attention, the kind of sound sources, and the condition of other circumstances. Hartmann.

GENERAL.

a.—REPORTS AND GENERAL COMMUNICATIONS.

274. VILLARET. The increase of ear disease in the German army. *Deutsche militärärztliche Zeitschrift*, 1899.

275. LAUFFS. On the results of aural treatment in deaf-mutes. *Medic. Correspondenzbl. d. Würt. ärzt. Landesvereins*, Nos. 40–43, 1899.

276. VÖLCKER. Arrested development of the speech centre. *Brit. Med. Journal*, Dec. 26, 1899.

274. In the twenty-three years from 1873–96, the number of aural affections in the German army which came under treatment has steadily increased, and has risen from 6.28 °/₀₀ to 12.12 °/₀₀. The increase was gradual and equal in all battalions. In addition to this increase, a decrease in mortality after ear disease was also noted. The number of discharges as unsuitable or invalidated on account of ear disease has considerably increased. The author is unable to give a cause for this increase, and refutes, according to the reviewer, without convincing proofs, the natural conclusion that the increase of ear disease is only apparent and due to the better otological knowledge during the last decade.

KÖRNER.

275. The patients were 59 deaf-mutes and one hearing-mute, and varied from 7–22 years in age. Examination revealed naso-pharyngeal adenoids to be the most frequent anomaly (61 %) ; also atrophic rhinitis and retractions of drum-membrane were frequent, and chronic aural suppuration, and simple opacity and perforation of the drum-membrane were rarer conditions. In 48.2 % of the congenitally deaf and in 23.6 % of those becoming deaf later, hearing of vowel sounds was preserved ; 35 % of the congenitally deaf and 81 % of the others were totally deaf for speech.

In 80 of the 120, various operations were undertaken ; removal of cerumen, adenectomy, tonsillotomy, etc. After the course of a few months, the excellent result was obtained that 49 were not improved but 37 were more or less improved, and it was found that in the congenitally deaf a disease of the sound-conducting apparatus or of the naso-pharynx was present in greater proportion than in those who became deaf, and further treatment in the former variety gives much the better hope of improvement.

In two cases, hearing necessary for ordinary life was obtained by removal of adenoid vegetations. The latter operation in four

other cases was followed by marked improvement. The author closes with the plea that children during their first year in a deaf-mute institute should be tested for their hearing and an appropriate treatment of the ears, nose, and throat be instituted.

MÜLLER.

276. At a meeting of the Clinical Society of London held on December 8th, VÖLCKER showed a girl aged seven and one half years, who was unable to speak. She was the elder of two children, her brother being healthy. The father's sister had a child, aged eleven years, who was said to be similarly affected, the family history being otherwise good. The child had been quite healthy until the age of six months, when she had a series of general convulsions. These fits recurred occasionally up to the age of three years, when they disappeared. She walked at twelve months, but had never spoken. She was quite rational and intelligent. The hearing was normal. Spontaneous speech was limited to a few monosyllabic sentences. She could not recognize printed or written words, numerals, or letters, but recognized pictures of objects or objects themselves. She was unable to write letters, words, or numerals, or to copy them. She could, however, copy straight lines or circles with either hand, preferably with the left. When writing with the left hand, she frequently made marks from right to left. Accepting the existence of a visual and an auditory perceptive centre and a glosso-kinæsthetic and cheiro-kinæsthetic centre as maintained by Bastian, Völcker thought that it appeared as if the two former centres were intact, but that the latter, or their commissural connections with the first two centres, were involved. He thought that the convulsions had in some way damaged the region referred to, and produced arrested development. The prognosis was thought to be favorable and the treatment recommended was instruction in writing and in lip language. ARTHUR H. CHEATLE.

b.—GENERAL SYMPTOMATOLOGY AND PATHOLOGY.

277. TODD, FRANK C. Conveyance of infection through the medium of the ear syringe. A remedy. *Jour. of the Amer. Med. Assoc.*, Oct. 14, 1899.

278. OPPENHEIMER, SEYMOUR. The effect of atmospheric changes on the hearing in chronic catarrhal otitis media. *N. Y. Med. Jour.*, Oct. 21, 1899.

279. MASIP, J. A. Otitis media in atrophic rhinitis. *Revista de Ciencias Médicas*, Oct. 19, 1899.

277. An ear syringe to be aseptic and practicable must meet the following requirements: 1. The point which comes in contact with the ear must be capable of sterilization and so constructed that it can be easily removed. 2. There must be no suction through the point.

The fountain syringe answers these requirements. Small glass points are used and changed after using in a septic case. The objection that the current of the fountain syringe cannot be regulated at will is overcome by a bulb attachment, which has a valve at each end. The solution is drawn in at one end and through a tube so large that the bulb is quickly filled. The rubber tube at the other end is smaller and terminates in a joint fitted with a shield to protect the operator from the return flow. The point can be unscrewed and sterilized. J. B. CLEMENS.

278. The observations and conclusions drawn are from a study of fifty (50) consecutive cases of chronic sclerosis of the middle ear, extending over a considerable period of time. The usual tests were used to determine the variations in the hearing under different atmospheric conditions.

Conclusions: I. The hearing in at least seventy per cent. (70 %) of the cases with chronic catarrhal deafness becomes worse under adverse weather conditions.

II. The degree of impairment of audition, as influenced by atmospheric conditions, is determined, to a great extent, by the location and the character of the pathological process in the tympanic cavity.

III. The morbid alterations most susceptible to barometric variations are those of hyperplasia.

IV. In purely atrophic changes in the middle ear, weather variations have little or no effect upon the auditory function.

V. Atmospheric influences also impair the hearing by unfavorably affecting catarrhal processes of the upper respiratory tract and Eustachian tube.

VI. All things being equal, the impaired audition in chronic catarrhal otitis is diminished more (under unfavorable conditions) in those whose general health is below par than in those otherwise healthy. J. B. CLEMENS.

279. MASIP arrives at the following conclusions from a study of nineteen reported cases:

1. Sclerosing otitis media developed in patients with atrophic rhinitis with considerable frequency — in one sixth of the

cases, presumably in direct connection with the nasal affection. The middle-ear inflammation forms a well-characterized group in the heterogeneous group of middle-ear scleroses.

2. These otitides are peculiar on account of the age at which they appear in children and young individuals; they occur about the same time in both ears with slight intensity without the paracousis of Willis (?), without labyrinthine symptoms or hyperæmia of the malleus or Shrapnell's membrane.

3. Some of the scleroses do not appear until at a later age; they are, however, to be regarded as the continuation of previous otitides.

4. Patients with atrophic rhinitis may be affected by other kinds of otitis and with greater frequency, independent of the nasal atrophy, as with acute or chronic catarrhal or even purulent otitis. HARTMANN.

c.—METHODS OF EXAMINATION AND TREATMENT.

280. MATTHAEI. Athletic respiration, a hygienic therapeutic aid in diseases of the nose, throat, and ears. *Therap. Monatshefte*, 1899.

281. STURROCK, CHARLES A. A method for the removal of foreign bodies from the nose and ear. *Brit. Med. Jour.*, Nov. 25, 1899.

280. MATTHAEI means by athletic respiration deep respiration with closed mouth for an hour to the full limit, with subsequent holding of breath for about fifteen seconds. Chronically inflamed mucous membranes, especially of the Eustachian tube, are thereby diminished. KILLIAN.

281. STURROCK applies suction by means of a piece of india-rubber tubing, rather less in diameter than an ordinary lead-pencil, and varying in length from one to three inches, according to the distance of the foreign body from the surface, attached to the nozzle of a brass syringe. He finds it advantageous to dip the tubing into glycerine, thereby diminishing the chance of air entering between the tube and the foreign body.

ARTHUR H. CHEATLE.

EXTERNAL EAR.

282. LERMOYEZ. A case of menstruation from the right ear. *Ann. des mal. de l' or., du lar.*, 8, 1899.

283. SCHIMANOWSKI. Paralysis of the abducent nerve following acute diffuse inflammation of the external meatus. *Westnik oftalmologii*, Jan., 1899.

282. A girl, fourteen years old, otherwise healthy, and who had never menstruated, suffered from hemorrhage from the right ear in monthly periods, after preceding feeling of malaise, which continued for several days. After three years, normal menstruation set in, though frequently accompanied by bleeding from the right ear and epistaxis. The ear canal was hyperæsthetic and presented some dilated vessels. ZIMMERMANN.

283. A few weeks after the aural affection, the paralysis set in and slowly disappeared with the healing of the ear canal.

SACHER.

MIDDLE EAR.

a.—ACUTE OTITIS MEDIA.

284. VOGT. Facial paralysis during acute otitis media. Heidelberg, *Inaug. Dissertation.*

285. LEWIS, ROBT. A brief history of five cases of mastoiditis. *N. Y. Med. Rec.*, Oct. 28, 1899.

286. TANSLEY, J. O. Shall we use cold in acute middle-ear or mastoid affections; if so, how long? *Laryngoscope*, Nov., 1899.

284. A complete exposition of the anatomical relations of facial nerve canal and description of the causation and clinical symptoms of facial paralysis, based on twenty-three cases collected from the literature and two observed at the Heidelberg clinic.

BRÜHL.

285. The writer's object in reporting these cases is to demonstrate the rapid and insidious development of serious complications in acute middle-ear inflammation, and to illustrate that the mastoid operation is, *per se*, unattended with danger.

CASE I.—A boy, aged nine, had otitis media acuta following scarlet fever and nasal diphtheria. The inflammation first attacked the right ear. Notwithstanding paracentesis of a bulging drum-membrane by Dr. Albert Buck, and douches of bichloride solution 1:6000, mastoiditis was well developed two days after. Operation showed the mastoid to be much involved, the cells filled with a quantity of pus. A few days after the mastoid operation the lymphatic glands in the neck of the same side suppurated; they were opened freely and much necrotic tissue

removed. Still later the left ear became inflamed. Mastoiditis followed, for which operation was performed. The patient's condition was poor, but it improved after each operation. Three days after the left mastoid was opened pericarditis and endocarditis, with mitral regurgitation and aortic obstruction, were discovered. The ears healed, but the patient died later of the cardiac involvement.

CASE 2.—The patient, a male, while apparently convalescent for two weeks from an attack of tonsillitis, suddenly developed otitis media acuta. Without any known cause, he was found, two days after, in a state of marked and alarming collapse. Membrana tympani red and bulging, especially in the posterior superior quadrant, in which was a small perforation, allowing the escape of pus. No tenderness, œdema, or redness over mastoid Temperature, 103.2° F.; pulse, compressible, intermittent, 120. Patient's condition critical. The mastoid cells were opened; bony walls were eroded and the large cavity filled with pus. Sigmoid sinus was exposed and found covered with granulations. A fistulous opening through the tympanic roof was found, though the overlying dura was healthy. Shortly after the mastoid operation, a phlebitis of the left leg and perihepatitis developed, prolonging the convalescence. The aural lesion finally healed.

286. TANSLEY, in reviewing the question and reporting a case in detail, reaches the conclusion that the use of cold in mastoiditis is more harmful than beneficial. Its application quiets pain and keeps down external swelling, thus masking the condition prevailing beneath, in the substance of the mastoid. Thus, as cold applied according to the prevailing methods is insufficient to destroy the microbes, its use should be discontinued. Early operation is urged, particularly if the middle-ear trouble is, or has been, an attical one. CLEMENS.

b.—CHRONIC OTITIS PURULENTA.

287. CIMA, F. Acid bacillus (smegma-bacillus) in the exudate of sucklings' otitis. *Archiv. ital. di Otol.*, vol. ix., p. 72.

288. PAUTET. Cholesteatoma of the ear. *Gazette hebdom. de médic. et de chirurg.*, No. 99, 1899.

289. VON ZUR MÜHLEN. A case of necrosis of the labyrinth. *St. Petersburg. med. Wochenschr.*, No. 13, 1899.

290. HESSLER. Middle-ear suppuration and cerebral tumor. *Arch. f. Ohrenhl.*, vol. xlviii., p. 36.

291. BARATOUX. The indication for the exposure of the middle-ear cavities in the chronic suppurations. *Le progrès médical*, Nov. 18, 1899.

292. LUCAE. Profuse escape of cerebro-spinal fluid for five weeks without cerebral symptoms. *Berl. klin. Wochenschr.*, No. 40, 1899.

293. LOMBARD. Essay on the indications of the opening of the mastoid process and of the middle-ear cavities in chronic purulent otitis. Paris, G. Steinheil, 1899.

294. TRAUTMANN. The persistent retro-auricular opening after the radical operation and the plastic closure of the same. *Arch. f. Ohrenhlk.*, vol. xlviii., p. 1.

295. HAMMERSCHLAG. The operative exposure of the middle-ear cavities in chronic otorrhœa at the University clinic of Professor Politzer. *Wien. klin. Wochenschr.*, No. 43, 1899.

296. KÜSTER. Osteoplastic exposure of the mastoid process. *Centralblatt f. Chir.*, No. 43, 1899.

297. PASSOW. Küster's osteoplastic operation on the mastoid. *Münch. med. Wochenschr.*, No. 49, 1899.

298. PANSE. On Professor Küster's osteoplastic operation on the mastoid. *Centralblatt f. Chir.*, No. 50, 1899.

299. KÜSTER. The criticism of Dr. Panse. *Ibid.*, No. 52, 1899.

287. Based on eight observations, CIMA reports that occasionally a bacillus resistant to acids can be found in the discharge of chronic purulent otitis, which resembles the tubercle bacillus but is classed among the smegma-bacilli. The tubercle bacillus is not so frequently found as is sometimes stated. A certain method to decolorize after treatment with carbol fuchsin is the use of acidulated alcohol for ten minutes. GRADENIGO.

288. PAUTET describes the clinical and pathological pictures of cholesteatoma of the ear. He supports the Bezold-Habermann theory of the origin of cholesteatoma and agrees with Siebenmann's views. SCHWARDT.

289. A poorly nourished child of two and a half years has had bilateral otorrhœa for one year after scarlet fever. The right canal is filled with polypi. Facial paralysis. Radical operation. The mastoid process was normal externally; the antrum, middle ear, and aditus filled with granulations; no ossicles found, all diseased parts removed. A regular after-treatment was not possible.

Four months later there was a fistula behind the ear, gangrene of the skin over two square centimetres, very fœtid discharge, exuberant granulations on the promontory, with area of white bony surface rough to the touch. After detachment of the auricle a large piece of bone and an entire circular canal were removed with the sharp spoon. This fragment of bone contained all the bones of the cochlea and the vestibule and the int. auditory meatus. Healing by aid of skin grafts. HARTMANN.

290. HESSLER reports eighteen cases of brain tumor occurring with chronic otorrhœa and adjoins a personally observed case. A girl, aged eleven, after scarlet fever, left acute otitis media, right deafness with no change in drum. The trouble in the left ear was complicated by a mastoiditis which necessitated operation. This was followed by occasional fever, vomiting, apathy. Considerable albuminuria. On the eighteenth day two transient convulsive seizures of the left, then of the right side, with unconsciousness, fever. Trephining, the dura appeared tense and was incised, negative puncture of the brain. Some improvement until the thirteenth day, severe pains were felt in the left ear, followed by coma, continuing to death. The trephine opening was again exposed; after release of necrotic brain matter two spoonfuls of clear cerebro-spinal fluid were discharged from a fistula which led to a cavity as large as an apple. Death four days later. At autopsy a sarcoma of the size of a child's kidney was found in the left temporal lobe.

Uræmia, brain abscess, or serous meningitis has previously been suspected. The presence of the tumor explains the right-sided, total deafness; this and the distended left lateral ventricle had completely flattened the left upper temporal convolutions.

In the complete discussion on the diagnostic difficulties of these rare cases, Hessler says that brain tumor must always be suspected and even hysteria. "The more certain the diagnosis, the surer are generally the results of operative treatment."

BLOCH.

291. BARATOUX considers the indications for complete exposure of the middle-ear spaces under the following headings:

1. In case of complicated otorrhœa.
2. To cure chronic purulent otitis media.

Under 1, it is necessary to make the complete exposure in presence of beginning meningeal symptoms. The dura is exposed and the wound thoroughly cleansed. The cerebral signs then

often disappear. If this is not the case after twenty-four hours, sinus thrombosis or brain abscess is present, and appropriate operative measures must then be undertaken.

The broad exposure of the ear spaces and the dura is indicated also when the brain symptoms appear in the picture of so-called "meningisme," *i. e.*, chronic irritative meningeal symptoms without any violent outbreaks.

Baratoux mentions MacEwen's observation that in acute exacerbations of a chronic otorrhœa, pneumonic attacks may occur which also call for the radical exposure.

The radical operation is indicated in well-marked subjective symptoms, fistula, facial paralysis, protrusion of the upper part of the ear canal, fungous granulations springing from the dura, and in local tuberculosis. Ossiculectomy and exposure of the attic are indicated in cholesteatoma, granulations, polypi, and perforations which have resisted non-operative treatment. In case of a relapse, the radical operation is then indicated.

SCHWENDT.

292. The seventeen-year-old patient was operated on by LUCAE because of an otorrhœa continuing after opening of the mastoid process. After opening the mastoid process a large bony defect of the size of a five-cent piece was found at the upper and posterior part, where the dura lay bare, and covered by a sequestrum measuring a square centimetre. On removing the sequestrum an opening was found in the dura and arachnoid which immediately discharged so large a quantity of cerebro-spinal fluid with blood as to bring the operation to an end. This discharge continued for five weeks, and during the first fourteen days necessitated a twice daily change of dressing. During all this period no cerebral symptoms whatever appeared. Lucae thinks this excessive production of cerebro-spinal fluid was caused by the irritation exerted by the sequestrum. MÜLLER.

293. A very thorough monograph (113 pp.) on this subject, with thirteen personal observations. HARTMANN.

294. TRAUTMANN repeatedly advocates the permanent retro-auricular opening. Thereby is avoided the deformity in the auricle caused by Siebenmann's plastic. The horizontal incision of the canal divides the flaps for the posterior surface into a narrow upper and a broad lower one, the vertical incision being made at, and not in, the concha. The lower flap is sutured to the inferior angle of the wound, while the upper is tamponed. To

hasten retarded epidermization, occurring, according to Trautmann, in long-standing otorrhœa, swollen and hyperæmic mucous membrane, chronic naso-pharyngitis, syphilis, tuberculosis, skin grafts are employed. To retard hypertrophy of the epidermis in healed cavities, a white precipitate salve, one per cent., is used, and dry sterile gauze is introduced. Membranes are sometimes formed in the situation of a regenerated drum; these, however, do not spring from remnants of the drum.

The retro-auricular wound diminishes with time. A year should pass before an attempt be made to close them, never in cholesteatoma. The closure of the epidermized fistula is done after Passow's method with some modifications. The auricle is then so placed that the scar is invisible. In the twenty-three cases reported, primary union always took place. The hearing was sometimes improved, sometimes made worse; the former appeared to be the case when the epidermis covering was thin. BLOCH.

295. In addition to the usually accepted indications for exposing the middle-ear spaces, POLITZER gives the following: 1. In obstinate suppurations from the antrum with fistula in the post-upper quadrant, especially when adhesions bind the drum remnants to the inner tympanic wall. 2. The discharge of gritty cholesteatomatous masses. 3. Otorrhœa with symptoms of beginning pulmonary consumption. The statistics embrace sixty cases. The indication, duration of after-treatment, and results as to healing are given. Finally the origin of endocranial otogenic complications is discussed. POLLAK.

296. KÜSTER has practised the so-called osteoplastic operation on eight patients. A tongue-shaped flap is made behind the ear, commencing above behind the ear down around the mastoid tip, and ascends along the posterior border of the mastoid down to the bone; the periosteum is elevated along the border and a thin bone plate adherent to the skin and periosteum is chiselled free; the flap is turned up and the mastoid is opened according to the method described by Küster in 1889. The flap is replaced and sutured except below, where a piece of bone is removed from the bony plates to permit gauze drainage. Advantages of the operation: No deformity except a thin scar, more rapid healing, finally safety of the antiseptic tamponade in injury to the sinus or dura, restoration of the bony outline, and in place of the deep furrow usually remaining after the ordinary opening there is a well-formed bone. The difference is so great that any one com-

paring the results of the two methods will not have any doubt as to the value of the osteoplastic method. Nine case histories and one illustration are added. BRÜHL.

297. The impracticability of Küster's method is demonstrated step by step in a very lucid and objective manner. The method described in 1889 suffers from an incomplete exposure of all the middle-ear cavities, and from the impracticable narrowness of the newly formed wound canal. The so-called radical method obviated these difficulties where the cavity was not allowed to fill with granulations but was clothed with skin. The new method, the osteoplastic, rests on the mistake that a deformity always follows the radical operation, while the opposite is the case, as, with aid of the plastic procedures, the wound can be sutured at once, which leaves only a thin scar. Granted that the osteoplastic method leads to cure, it would if accepted mean a loss of ten years' labor in otology. In full recognition of Küster's merits the author concludes as follows, in which he has the support of all otologists: "I have well considered the matter before I opposed the views of the meritorious Marburg surgeon. I consider it my duty, for if Küster's suggestions are accepted, completely wrong impressions on the value of the radical operation will arise."

BRÜHL.

298. PANSE opposes Küster's proposition and says it means such a marked step backward that it cannot be too soon warned against because (1) the technic is bad (one facial paralysis, injury to the sinus); (2) the result is cosmetically inferior to Stacke's or Panse's plastic. Healing did not take place in one third of the cases.

299. KÜSTER's reply to the preceding, without furnishing any new features to the question. BRÜHL.

c.—CEREBRAL COMPLICATIONS.

300. GRADENIGO. On the diagnosis and curability of otitic leptomeningitis. *Arch. f. Ohrenhlk.*, vol. xlvi., p. 155.

301. MÜLLER, R. On the operative treatment of otitic meningitis. *Deutsche med. Wochenschrift*, No. 45, 1899.

302. FERRERI. Severe peri- and endocranial complication after acute otitis running a chronic course. *Arch. ital. di Otol.*, vol. ix., p. 49.

303. KIRMISSON. Cerebral abscess. *Le progrès médical*, Nov. 18, 1899.

304. KADJAN. Abscess of the temporal lobe of the brain. Letopiso russkoi chirurgii. Two autopsies. *Journ. Am. Med. Assoc.*, Nov. 11, 1899.

305. JÜRGENS. Streptomycosis of the ear. *Monatschr. f. Ohrenhlk.*, 1899, No. 11.

306. YOUNG, ARCHIBALD. Remarks upon the operative treatment of infective thrombosis of the sigmoid sinus following chronic purulent otitis media. Record of case successfully treated. *Glasgow Med. Journ.*, Oct., 1899.

307. SCHRAGA. Sinus-phlebitis from chronic otitis; operation; recovery. *Monatschr. f. Ohrenhlk.*, No. 10, 1899.

308. MEIER, E. Otitic pyæmia. *Münchn. med. Wochenschr.*, No. 43, 1899.

309. RANDALL, B. ALEX., and ADAMS, JEANNIE S. Lateral sinus-phlebitis after otitis media in typhoid fever. *University of Pa. Med. Magazine*, Dec., 1899.

310. RANDALL, B. ALEXANDER. Four cases of cerebellar abscess. One success. Two autopsies. *Journ. Am. Med. Assoc.*, Nov. 11, 1899.

300. GRADENIGO reports four cases.

I. Chronic bilateral purulent otitis since childhood, right perisinuous abscess, and beginning thrombosis of transverse sinus; death from basilar meningitis. A girl fourteen years old, jaundiced when admitted to the hospital, hard of hearing for one year; the left canal was occluded by a polyp. From the twentieth day after the radical operation constant fever, headache, vomiting, nystagmus, stiff neck, facial paralysis on the right side, delirium, and coma. The autopsy showed the above condition on the right side. There was pus in the right int. auditory meatus. Gradenigo believes that the concussion of the operation on the left ear may have produced the lesion on the other side.

II. Woman, twenty-seven years old, with left-sided otorrhœa since youth, and transient facial paralysis. Recurring polypi, meningeal symptoms. Operation: empyema of antrum; the sinus was exposed but appeared healthy. On probing in an upward direction considerable discharge of pus from an extradural abscess above the antral roof. After removal of the latter the dura of middle fossa was found covered with granulations and a purulent membrane. Operation interrupted. High fever, continuous meningeal symptoms. Second operation: the dura ex-

posed to healthy parts and the middle-ear cavities thoroughly exposed, removing cholesteatoma and granulations. Recovery.

III. Boy thirteen years old, acute otitis media in left ear for two weeks, high fever, meningeal symptoms. Mastoid process was normal. Paracentesis evacuated much pus. No improvement. Lumbar puncture: in the cloudy cerebro-spinal fluid leucocytes and very virulent staphylococci. Gradual recovery.

IV. A woman, thirty-five years old, right cholesteatoma, otorrhœa for nine years, fever during last two weeks and recently meningeal symptoms. Bulging of posterior and upper wall. Lumbar puncture showed a fluid with many white blood corpuscles and staphylococci. At operation cholesteatomatous masses were removed from antrum and middle ear. Dura free. Later facial paralysis. Ocular fundus hyperæmic. Nine days later fever disappeared and gradual recovery set in. Gradenigo believes the lumbar puncture to have a curative effect and that packing the wound with two per cent. carbolic gauze is very favorable.

BLOCH.

301. MÜLLER reports two cases of serous meningitis, of which the one is chronic externa and the other ventricular or acute interna.

I. Mening. serosa ext. chron. An otherwise healthy girl was taken ill in April, 1895, with mastoiditis, following ac. otitis media. Simple operation, healed at Christmas, 1895. Moderate headache and vertigo. In Sept., 1898, sudden aggravation of all symptoms, though the ear did not again suppurate. The radical operation exposed a large cavity, completely empty, with black, necrotic, absolutely dry walls in the mastoid process below the scar. An abscess in the temporal lobe was suspected and a number of punctures were made through the dura; no pus, but some serous fluid. Incision with a knife to a depth of 3 *cm* was also negative. A trephine opening was made through the squama. On opening the dura, a quantity of serous fluid escaped, but no pus. The escape of fluid continued during recovery. Two months later the brain wound was healed without any prolapse, and two months later the ear wound was also completely healed. A complete recovery has not, however, taken place, as vertigo, imperfect locomotion, reduced sensation of the crossed side, and the recent aggravation (increased headache, vomiting, tenderness on percussion of the left hemisphere, normal eye-ground, pulse, and temperature) persisted, though the operation on Sept. 15, 1898, can

be regarded as a life-saving procedure. These symptoms are probably due to a chronic inflammatory serous infiltration of the brain substance, especially of the temporal lobe, as a result of the non-purulent, necrotic disease of the mastoid process.

II. Mening. seros. interna acuta. Woman thirty-one years old, mother of three healthy children, was taken ill with meningeal symptoms suggestive of a brain tumor, though she had had otorrhœa for many years. The radical operation was performed on account of the otorrhœa and the tenderness of mastoid. The antrum contained cholesteatoma, but trephining of the temporal lobe proved negative. The general condition did not improve; after a few days the region over the cerebellum was trephined, but again no pus was found. No improvement. An enormous cerebral prolapse appeared at both openings. Four weeks later, a gradual improvement of all symptoms began with a serous transudation of the bandage; complete recovery in three months. The diagnosis of meningitis serosa interna, with exudation into the ventricles, was made by exclusion, and puncture of the ventricles is advocated in this and similar cases. No brain symptoms remained, notwithstanding the great loss of brain tissue. The author believes that the trephine openings should be made away from the wound of the radical operation, to guard against infection of the brain hernia by the otorrhœa. NOLTENIUS.

302. Report of two cases with severe complications without revealing any involvement of the mastoid processes. It is supposed that in both cases the tubal cells described by Bezold were affected and caused the deep abscesses in the neck and pharynx. Most of the symptoms were referable to the deep parotid region. The operative treatment of analogous cases is discussed.

A case of extradural abscess after acute otitis media without mastoid disease is also described. The author is in favor of operation through the ear. GRADENIGO.

303. KIRMISSON showed a patient before the Paris Surgical Society on whom he had operated on account of brain abscess. The pus contained streptococci; healing was uneventful, but the half-sided paralysis and contracture remained for some time. The contracture and paralysis disappeared almost completely with massage. SCHWENDT.

304. Acute suppurative otitis after follicular tonsillitis. One month later mastoiditis, and at the same time the characteristic

symptoms of an abscess in the temporal lobe. Operation. Healing. SACHER.

305. A soldier who had had a chronic purulent otitis and mastoiditis succumbed to a thrombosis of the left transverse sinus, meningitis, rapid softening in the temporal lobe, and septicæmia. Pure cultures of streptococci were found in the softened areas of the brain, in the antrum and mastoid cells, labyrinth, and middle ear. KILLIAN.

306. YOUNG's case was that of a child two and a half years of age, the chief interest being that recovery took place without ligation of the internal jugular vein, the sinus being incised and the septic contents turned out. The thrombosis occurred high up, at the knee of the sinus. ARTHUR CHEATLE.

307. At the operation the sinus was found surrounded with pus. It was opened three days later, when distinct pyæmic symptoms had supervened. Ligation of jugular vein. The sinus contained soft, brownish-red masses of thrombus. Later several pyæmic abscesses had to be opened. Recovery. KILLIAN.

308. Report of eight cases of pyæmia, all due to sinus thrombosis ; of these three had previously been reported. In all cases operation was performed ; twice the jugular was ligated. In three the otitis was acute ; one of these was fatal. In four the otitis was chronic, with a mortality of two. MEIER agrees entirely with Leutert's explanation for the development of pyæmia.

SCHEIBE.

309. In this case, after three months of malaise, typhoid fever developed which was later complicated by the occurrence of middle-ear inflammation. The otitis never became purulent in character in the right ear, though reported at times puriform in the left ear. During the course of the fever, superficial furuncular abscesses formed over the sacrum and shins, but no rigors, sweating, or characteristic septic temperature were noted. Some ten days after the crisis a relapse occurred, the pyrexia beginning with the only approach to a rigor observed. Later, while apparently convalescent for a period of two weeks, clear signs of an inflammation of the right mastoid and lateral sinus developed, which disappeared in three days without operation and was followed by an uninterrupted recovery.

CLEMENS.

310. CASE I.—The patient, fifteen years of age, had a discharging ear for three years. A box on the ear three days pre-

vious to the consultation was followed by nausea, pain, and malaise. The mastoid was red, tender, and swollen; marked fluctuation, temperature 106° F., appearance anxious and serious. Immediate operation refused. Mastoid opened next day. Incision through soft parts evacuated two drams of pus, surface of bone intact. Mastoid sclerosed, pus found with little caries when antrum was reached. No sinus was detected leading to adjoining parts. Antrum curetted and packed. Temperature fluctuated, eye-ground normal. With a temperature of 103° F., pleural friction was detected with rapidly following effusion and lung consolidation. No rigors, no jugular tenderness, no swelling about the neck. Patient died six days after operation and thirteen days after the injury.

Autopsy, twenty-four hours after death, showed amazing destruction of both lungs, pleural empyema, dura engorged and adherent, pia clouded. Two drams of pus evacuated upon removing the brain, owing to cerebellar abscess. Dura intact. Doubtful evidence of phlebitis. Cerebellar abscess size of pigeon's egg, with thick pyogenic membrane, thinnest in proximity to the antrum. Abscess may have antedated the injury.

Case 2.—Patient fourteen years of age. Had suppuration for two months; granulations found over the posterior wall of auditory canal. Operation showed the posterior osseous wall largely destroyed, mastoid one large cavity, sigmoid sinus large and somewhat forward, bony covering destroyed for one inch and covered by granulation. Considerable caries of inner plate. Slight subdural pus; two square inches of the dura exposed. Healthy bone was reached in all directions during the operation. The patient's general condition was bad, but after ten days was discharged and attended the clinic. Two weeks later, an abscess formed at the clavicle which was evacuated and a sinus was followed upwards for two inches. Healing occurred rapidly below but oozing from mastoid sinus continued. The appetite failed, with occasional vomiting, other serious symptoms following. With a sub-normal pulse of seventy-six (76), temperature of 98° F., cerebellar abscess was expected and operated for. The operation extended deep into the middle and cerebellar fossæ. An accident to the mastoid vein by rongeur prevented further exploration. Patient died. No autopsy.

Case 3.—Child of six years of age with middle-ear suppuration. The mastoid was opened, carious bone and granulations

removed. At a dressing of the wound a rough, overhanging edge of the cortex was scraped smooth and a small bone sinus found, which led to an abscess cavity about one *cm* in diameter, in the cerebellum. Healing was exceedingly slow, but the patient was ultimately discharged cured.

CASE 4.—A child four years of age. Suppuration of the right ear followed by mastoid abscess, which had been incised three weeks before case came under observation of writer, leaving a discharging sinus behind the ear. Much headache on the right side. Condition became serious later, with vomiting, convulsive twitchings of left arm and leg, without paresis. Little mental disturbance. Temperature 98.4° F. Respiration 20. Pulse 88. Mastoid was opened, carious bone and granulations removed, and, as no defect of the inner table could be detected, further operation was delayed. Death.

Autopsy: An excess of fluid in the arachnoid and ventricles was found. Cerebellar abscess about 3 x 5 *cm* occupied nearly the entire lobe. J. B. CLEMENS.

d.—OTHER MIDDLE-EAR AFFECTIONS.

310*a*. ARSLAN. Several syphilitic varieties of the cartilage of the Eustachian tube. *Arch. ital. di Otologia*, etc., vol. ix., p. 9.

311. STUCKY, J. A. Fractured base, with deafness, tinnitus, exophthalmus, facial paralysis, mastoiditis. *Jour. Am. Med. Assoc.*, Nov. 11, 1899.

310*a*. ARSLAN presents observations of a clinical picture of hyperplasia of the mucous membrane and the cartilage of the Eustachian tube in tertiary or hereditary syphilis. The most constant symptom is the loss of hearing from tubal stenosis; this does not correspond to increase in volume, which is not recognizable in the rhinoscopic examination; it is probably produced by the extension of the disease to the walls of the tube. Specific treatment gave the best results. GRADENIGO.

311. Case of a jockey, twenty-one years old, who was thrown from a horse and sustained a large contusion of the scalp over the vortex, which was rapidly followed by œdema, extending down to both ears, though more marked on the right side. Tinnitus and deafness in right ear, no hemorrhage from nose and throat. Five weeks after the injury exophthalmus of right eye developed, hemorrhagic spots in deep conjunctiva, dimness of

vision, eye-ground negative. Complete facial paralysis, swelling of mastoid integument, tenderness over antrum and tip of the bone. Auditory canal red and swollen, bulging posterior superior wall. Perforation of the drum, in the superior posterior quadrant, and discharging offensive pus. Constant headache; vertigo, falling toward the left; aphasia.

A Stacke-Swartze operation was performed, and the attic, middle ear, antrum, and cells found full of firmly adherent clots: no pus; no caries. Malleus was found separated from the drum and incus from the stapes. The clots and inflammatory products were thoroughly removed, cavities cleaned, and the mastoid dressed in the usual way. All symptoms but the facial paralysis disappeared after the operation. Recovery was uninterrupted.

J. B. CLEMENS.

NERVOUS APPARATUS.

312. LANNOIS, E., and HARDOUR, M. On true hysterical deafness. *Ann. des mal. de l'oreille*, etc., No. 10, 1899.

313. ALT. On psychic deafness. *Monatschrift f. Ohrenheilk.*, No. 12, 1899.

314. FERA. A case of monolateral multiple progressive paralysis of the cranial nerves. *Arch. ital. di Otol.*, etc., vol. ix., p. 34.

312. LANNOIS and HARDOUR endeavor to separate the clinical pictures of hysterical deafness from other affections which are somewhat similar. The following should not be confounded with the true hysterical deafness: (1.) Hysterical deafmutism; (2.) inattention of deaf persons due to psychic depression (désperance auditive). The deaf one, discouraged by his unsuccessful attempts to hear, gives up all efforts.

True psychic deafness is complete or nearly complete. It occurs without a material change being present in the ear, and forms the principal symptom of a general neurosis. Recovery follows spontaneously or after a psychic treatment. The author describes two cases of male hysteria. The main symptoms are the following: 1. The degree of deafness is greater than that associated with middle-ear diseases; the deafness corresponds to labyrinthine or nerve deafness. 2. Bone-conduction is abolished. 3. The drum membrane appears normal. 4. Insuction of the drum membrane is inconstant. 5. The disease is of equal intensity on both sides. 6. Subjective symptoms are of only short

duration. 7. Usually other symptoms of general hysterical neurosis are present, anæsthesia, contraction of visual field, etc. 8. A radical cure follows. SCHWENDT.

313. An historical and critical discussion on the above theme. KILLIAN.

314. Man fifty years old, with complete left-sided facial paralysis with pains and tactile anæsthesia of the left half of the face. Loss of hearing, left, due to the affection of sound-conducting and nervous apparatus. Complete paralysis of the left vocal cord. Later, necrosis of the left cornea and movements of deglutition. Death from inspiration pneumonia. At autopsy a sarcoma of the left middle fossa of the skull, extending to the anterior and inner part of the posterior fossa, to the left half of the sphenoidal cavity, and the posterior end of the middle turbinal. The tympanum was free. The left basal nerves are compressed and invaded with tumor masses. GRADENIGO.

NOSE AND NASO-PHARYNX.

a.—GENERAL PATHOLOGY.

315. REUTER. Essential anosmia. *Arch. f. Laryng.*, vol. ix.

316. PLACZEK. Congenital absolute bilateral anosmia. *Biol. klin. Wochenschr.*, No 51, 1899.

317. FRÄNKEL. Open mouth and short upper lip following shortening of frenulum labii superioris. *Arch. f. Laryng.*, vol. ix.

318. CORDES. Mucoid degeneration of the epithelium of glandular ducts in the nasal mucous membrane. *Arch. f. Laryng.*, vol. x.

319. ZUCKERKANDL. On the development of the concha bullosa. *Monatschr. f. Ohrenheilk.*, No. 10, 1899.

315. REUTER divides the essential anosmias with probable anatomical causation in three groups after their etiology: 1. The anosmia remaining after the complete extirpation of genuine nasal polypi; 2. The anosmia in chronic ethmoiditis; 3. The anosmia in ozæna. In the first class, while in many cases the smell returns after the removal of polypi, it may be permanently damaged. ZARNIKO.

316. A woman, sixty years of age, had never possessed the faculty of smell. There were no changes in the local condition nor in the nervous system. A similar case is described in a man

forty-four years of age. These two, besides a case of Zwaardemaker's, are the only ones on record. MÜLLER.

317. FRÄNKEL observes three successive cases of the mouth being kept open from abnormal shortness of the frenulum labii sup. The maxillæ and lips were normal. The deformity was permanently cured by simple division of the band with the scissors. He suggests the name of mikrocheila. ZARNIKO.

318. CORDES has studied the bud-like structures, first described by the reviewer, then by Birmingham and Okada, which occasionally occur in hyperplastic epithelium of the nasal mucous membrane. These are not independent mucous glands but belong to normal mucous glands caused by the mucoid metamorphosis of the cells surrounding the excretory duct in the epithelium. ZARNIKO.

319. The cavities in the middle turbinates may be continuations of the upper-middle meatuses or of a bulla cell or of an anterior frontal cell. There are usually one, and sometimes two or three of these cavities.

The paper of Bergeat (*Münch. medic. Wochenschrift*, 1897) seems to have escaped ZUCKERKANDL's attention. KILLIAN.

b.—METHODS OF EXAMINATION AND TREATMENT.

320. BOCK. Experiences with electrolysis, especially in nasal therapeutics. *Berl. klin. Wochenschr.*, No. 45, 1899.

321. BAUMGARTEN. Schleich's procedure in operations of the deviations and spurs of the septum. *Arch. f. Laryngol.*, vol. ix.

322. BREITUNG. The importance of the electric internal drum massage of the nasal mucous membrane for the general physician, and its technic. *Deutsche Medizinal-Zeitung*, No. 96, 1899.

323. BAUMGARTEN. The bloody treatment of hypertrophies in chronic rhinitis. *Wiener med. Presse*, No. 46, 1899.

320. BOCK's experience with electrolysis in ozæna are not encouraging; the method is, however, of value for cosmetic purposes (warts, nævi, calcified atheromata, etc.). It is especially serviceable in deformities of the septum, and, according to Brock, combines all the advantages of other methods and should be employed except in especially prominent traumatic deviations or where the necessarily prolonged treatment (six to seven weeks) is no objection. MÜLLER.

321. BAUMGARTEN operates on septum deformities with a chisel and painlessly from the use of Schleich's injections. Hem-

orrhage is also reduced, though it is more profuse later and requires careful tamponade. Schleich's method is also described in the division of synechiæ and in tracheotomy. ZARNIKO.

322. BREITUNG has modified the apparatus for vibratory massage, by which the action is made more uniform. This massage is supposed to correct all disturbances due to increased intracranial pressure, and to cure nervous coryza. It exerts a favorable action on ozæna and not only opens but keeps the ostia of the Eustachian tube open. HARTMANN.

323. BAUMGARTEN recommends removal of hypertrophies with the angular scissors without cocaine. POLLAK.

c.—OZÆNA.

324. COZZOLINO. A study of the bacteriology and histology of ozæna. *Ann. des mal. de l'oreille*, etc., No. 7, 1899.

325. PEWNIZKI. Treatment of ozæna with diphtheria antitoxin. *Wojenno medizinsky Shurnal*, Sept., 1899.

324. According to COZZOLINO, ozæna is due to a primary nutritive disturbance of the bony turbinals, to which is associated secondarily a bacterial infection. The latter is caused by the bacillus mucosus, which produces the fever and crusts.

ZIMMERMANN.

325. Three cases which received no other treatment than the serum injections. The results were absolutely negative. Electrolysis was also tried. After two or three sittings a complete cessation of the fœtor occurred. The method is, however, painful, and only temporary. SACHER.

d.—ACCESSORY SINUSES.

326. WROBLUOSKI. Acute empyema of the antrum of Highmore. *Arch. f. Laryng.*, vol. x.

327. RETHI. Negative air-douche as diagnostic aid in diseases of the accessory cavities. *Wien. klin. Rundschau*, No. 43, 1899.

328. GRÜNWALD. On the curability of inflammations of the maxillary antrum. *Arch. f. Laryng.*, vol. ix.

329. LICHTWITZ. Sequestrum developing about the operative canal, in the operative treatment of maxillary empyema through the alveolus. *Arch. internat. de lar., d'ot.*, xii., 4.

330. STRAZZA. Clinical remarks on the chronic inflammations of the frontal sinus, especially as to treatment. *Arch. ital. di Otol.*, etc., vol. viii., p. 361.

331. LUC. A case of unusually obstinate frontal empyema. *Arch. internat. de lar., d'otol.*, xii., 4.

332. CAUBET and DRUAULT. Meningitis and orbital abscess following a polysinusitis of dental origin. *Ann. des mal. de l'or., du lar.*, xxv., p. 8.

333. KOEBEL. Combination of otitis media with rhinogenic brain abscess. *Beiträge zur klin. Chirurgie*, xxv., 2.

334. LAFRANÇOIS. Ethmoid empyema with orbital complications. *L'année médical de Caen*, Sept. 15, 1899.

335. FERRERI. Fibrosarcoma of sphenoidal sinus. *Arch. ital. di Otol.*, vol. viii., p. 445.

336. GRUNERT. A new plastic method after complete exposure of the frontal sinus for empyema. *München. med. Wochenschr.*, No. 48, 1899.

337. KYLE, D. BRADEN. Confined suppuration of the frontal sinus with spontaneous rupture. *N. Y. Med. Jour.*, Dec. 16, 1899.

327. The use of the negative Politzer's experiment requires only a few seconds and often succeeds. The nose is first cleaned and dried, cocainization of middle meatus. Some water is held in the mouth, the nozzle of the compressed bag introduced in the nostril; during deglutition the bag is allowed to expand. It succeeds almost always in diagnosing accessory sinus disease. If no discharge appears, iodide of sodium is administered for two to three days to produce a profuse discharge, and the experiment is repeated. POLLAK.

328. GRÜNWALD has examined 106 cases with view to duration, character of secretion, condition in nose and of the teeth, and various complications. In general there is an inverse proportion between duration of disease and result of treatment. Not the catarrhal but the purulent forms are more favorable for healing; unfavorable are those with the ozæna complex (broad nose, crusts). Complications with polypi mean severe disease of the mucous membrane and make the prognosis worse. Contiguous diseased teeth make a permanent cure impossible. The prognosis is better if the tooth trouble is immediately recognized and treated. Some cures are prevented by diseased roots of normal-appearing teeth. Other remarks on the complication with suppuration of other accessory cavities, bilateral disease, the conditions within the cavity (polypi, polypoid excrescences,

diverticula) follow. Regarding therapeutic measures, the author does not think much of simple perforation, and employs it only where disease of the teeth or a defect at the corresponding place is present, in not too inveterate cases in young rather than old individuals, in catarrhal rather than purulent forms. Of operations with broad exposure he reserves Bönninghaus's method for the severest cases. Finally he emphasizes that many catarrhal diseases will get well by correcting the intranasal changes.

ZARNIKO.

329. The antrum of Highmore was opened from an alveolus with the electric trephine. Four weeks later, after pain had existed since the third day, an annular sequestrum was discharged. LICHTWITZ believes that the necrosis was due to overheating from too rapid moving of the trephine. ZIMMERMANN.

330. STRAZZA reports 5 cases and discusses the diagnostic features and treatment of chronic frontal empyema. Even though both sides were affected, the symptoms were only on one; the septum was always present, but softened in 2 cases and thinned in 1. In a certain number it is impossible to introduce a canula in the natural passage; even if successful, it is painful, and the curative action of irrigations is very small, especially as, in most cases, the sinus is filled with polypi. For radical treatment broad external exposure is recommended, so that the soft parts may fall back and the cavity be obliterated. No attention is paid to cosmetic reasons. It is necessary to remove all fungoid masses and the purulent focus. The author is against immediate closure of the wound and tampons until a granulating surface has formed. It is not necessary to pay any attention to the nasal duct. GRADENIGO.

331. A patient, twenty years old, had been operated on twice for frontal empyema with primary suture; the suppuration returned and extended to the other side. At the third operation both sinuses were exposed, the anterior ethmoid cells curetted, and the wound was again closed. Six weeks later fluctuation appeared over the left eye, which was opened and drained. The fistula closed after three weeks, though pus still collected. LUC put on a pressure bandage, with the result that at the next dressing the fluctuating had extended to the scalp limits. Several periosteal and an extradural abscess were formed, which was operated on. The patient finally died from meningitis. Luc has successfully operated on twelve frontal empyemata, and claims

that the failure in this case was due to constitutional peculiarities of the patient, for which there seem to be no reasons whatever.

ZIMMERMANN.

332. The sickness began like influenza, with coryza; then fever, vomiting, severe headache, painful œdema of the eyelids appeared. The incision of the lower lid evacuated a drop of pus, and no carious bone could be detected at depth. Death ensued after delirium. Autopsy showed a basal meningitis (right) especially in Sylvian fissure, produced by a small destruction of bone in the anterior part of the sella turcica. This led into the left sphenoidal sinus, which was distended to the right and filled with pus. The ethmoid cells and maxillary antrum were likewise affected, and into the latter projected a carious tooth. The ocular symptoms were caused by the transmission of the cavernous sinus and the ophthalmic vein. ZIMMERMANN.

333. A male, thirty-three years old, suffered from right chronic otorrhœa and bilateral purulent discharge from the nose; two weeks later 38.4°, vomiting, headache, vertigo, stupor, twitches in left arm. No tenderness over mastoid or forehead. Suspecting otitic brain abscess, trephine opening in squama, brain incised, no pus. Antrum opened, found filled with pus. Radical operation. Death after several hours. At autopsy, caries of posterior wall of right frontal sinus and abscess in right frontal lobes were found. Publication of hitherto reported cases (20) of rhinogenic frontal abscess and their symptoms.

BRÜHL.

334. A boy, four years old, fell on his nose and had headache for three days. Six months later, he was again taken ill with fever and became stuporous; left eyelid œdematous, slight exophthalmos. Examination of nose negative. As an osteoperiostitis of the inner orbital wall was suspected, an incision was made at level of inner canthus. A probe encountered ethmoid cells filled with pus. A broad opening was made between the ethmoid cells and the nose. Recovery. SCHWENDT.

335. FERRERI concludes as follows: a sphenoidal empyema should be diagnosticated as early as possible and operated upon, lest fatal intracranial complications follow. It is necessary to differentiate between a pyogenic inflammation and a neoplasm, as the same symptoms may be produced by either for a long time.

GRADENIGO.

336. After eradicating the frontal sinus by Kuhnt's method,

at both ends of the supraorbital horizontal incision, vertical incisions are made passing above and below. By undermining, two flaps are thus formed. The upper flap is placed in the frontal cavity after the epidermic layer has been removed, and the lower is pulled over this and sutured to it after being changed to a wedge shape. This method has been employed in one case with good cosmetic result. GRUNERT recommends his procedure only when the cavity is not too deep. SCHEIBE.

337. A woman, æt. sixty, experienced a fulness on the left nasal side, thin nasal watery discharge, swelling over the face, particularly between the eyes, and soreness at the inner angle of the left eye. The swelling increased, the nasal discharge became more pus-like, and malaise and general debility ensued. The patient had lost over thirty pounds in flesh. The swelling increased so much as to hang down over both supraorbital ridges, with marked swelling under both eyes. In the median line, an inch and a half above the line of the supraorbital ridge, was a tumor-like red projection, pitted in the centre with a small spot showing some dried secretion. In removing the dried crust an opening was found, and upon pressure foul-smelling, thick pus was discharged. The necrotic area was almost circular, ¾ inch in diameter. A probe passed easily through the nose. The outer opening closed spontaneously after two months. The necrosis had involved the outer plate only. M. TOEPLITZ.

e.—OTHER DISEASES OF THE NOSE.

338. LERMOYEZ. The treatment of nasal hydrorrhœa with atropine and strychnine. *Ann. des mal. de l'or., du lar.*, XXV., 7.

339. FREUDENTHAL. Excessive epistaxis controlled by local injections of gelatine. *Deutsche med. Wochenschr.*, No. 49, 1899.

340. FEDOROW. Forced dilatation of the chest as a means of checking epistaxis. *Bolnitschnaja gaseta Botkina*, No. 29, 1899.

341. COTTELL, A. B. Hemorrhage through the lachrymal duct after plugging the nares. *Brit. Med. Jour.*, Dec. 16, 1899.

342. GREEN, W. E. Case of rhinolith. *Brit. Med. Jour.*, Nov. 4, 1899.

343. MORF. Contribution to the etiology of the genuine fibrinous rhinitis. *Correspondenzblatt f. Schweizer Aerzte*, 1899.

344. HEINDL. On the treatment of rhinoscleroma or scleroma. *Ann. des mal. de l'or., du lar.*, XXV., 7.

345. MANASSE. On multiple amyloid tumors of the upper respiratory passages. *Virchow's Archiv.*

346. SEIFERT. Tuberculosis of naso-lachrymal canal. *Münch. med. Wochenschr.*, No. 52, 1899.

347. The relation of pathological conditions in the ethmoid region of the nose, and asthma. SWAIN, HENRY L., Pathology, *N. Y. Med. Jour.*, Oct. 28, 1899; RICE, CLARENCE C., Clinical Phases, *N. Y. Med. Jour.*, Nov. 11, 1899; BOSWORTH, F. H., Treatment, *N. Y. Med. Jour.*, Nov. 18, 1899.

338. LERMOYEZ has returned to the purely medical treatment of vaso-motor coryza; he recommends strychnine, atropine, āā 0.005 to 400 syr., 1–3 teaspoons daily. Of 27 patients treated in this manner 14 could be re-examined and 11 proved to be cured.

ZIMMERMANN.

339. A woman, sixty-eight years old, suddenly was taken with profuse epistaxis. Attempts at checking the bleeding with packing anteriorly and later posteriorly were only temporarily successful. The dangerous condition was avoided by an infusion of salt solution in the intra-clavicular subcutaneous tissue. The hemorrhage did not cease until 20–30 *ccm* of fluid-warm gelatine was injected into the nose with a warmed glass syringe.

NOLTENIUS.

340. FEDOROW has found the following method the best: the patient sits upright on a chair, places both arms on his head, and takes long, deep breaths with mouth open. SACHER.

341. A soldier who was much debilitated had a severe attack of epistaxis, necessitating plugging. As a result, blood appeared in each lower eyelid and trickled down the cheeks.

ARTHUR H. CHEATLE.

342. At a meeting of the Southern Branch of the British Medical Association, Isle of Wight Branch, held October 20th, GREEN showed a rhinolith which he had removed from a girl who had been troubled with her nose for years. The stone, which occupied the nose and naso-pharynx as far as the pharynx, was crushed with strong polypus forceps and removed in five or six pieces. Three months later, the symptoms having recurred, a thin ragged plate about 1 inch long, ½ inch wide, and ⅛ inch thick, was removed from near the back of the nasal cavity.

ARTHUR H. CHEATLE.

343. MORF collected all cases of fibrinous rhinitis from the literature and added three of his own. These are his conclusions:

Genuine fibrinous rhinitis is not to be distinguished etiologically, anatomically, or clinically from diphtheria, hence the same protective measures are required for either.

In the author's cases, virulent Löffler's bacilli were found in the pseudo-membranes. Recovery followed the use of antitoxin.

In several cases described by others, no Löffler's bacilli were found, but staphylococci and streptococci. According to the author, it is possible that the Löffler's bacilli were overgrown by the other cocci so that they no longer were present in the pseudo-membranes. SCHWENDT.

344. Of the eleven cases described, nine concerned the nose and naso-pharynx; in all cases typical rhinoscleroma bacilli were found. Treatment consists in restoring the nasal respiration with avoidance of destruction. The infiltrates and tumors were curetted, bands were divided and kept from reuniting by packing with gauze. ZIMMERMANN.

345. MANASSE has examined microscopically a case of infiltrating amyloid tumors of the larynx and trachea, and a nodular tumor in the right palate, tonsil, and larynx. BRÜHL.

346. Fourteen cases, of which thirteen belonged to the eye clinic. In five cases the tubercular process was localized to the lachrymal canal; in the other cases the conjunctiva or cornea was also affected. In all fourteen cases the nose was also involved. Secondary tuberculous inflammations of the lachrymal canal from the nose are the most frequent. SCHEIBE.

347. SWAIN holds that in asthma there must be first an irritability of the bronchial structures, secondly some other diseased organ, such as the nose, stomach, ovary, etc. And thirdly, the neurotic habit. The cause is found outside of the body, in certain irritations, as, *e. g.*, the pollens of grasses in hay fever, flour in baker's asthma, or in the musty smell of feathers, the last being illustrated by a case of a young man, æt. twenty-eight, whose asthma and even œdematous swelling of the middle turbinate disappeared after the change of his feather pillow. Swain then gives an elaborate theory upon the production of this œdematous tissue and how it produces asthma.

RICE believes that ethmoid diseases are not often associated with periodical asthma. The coexistence of asthma and ethmoidal disease is due to mechanical obstruction, necessitating mouth-breathing, and to the supervening chronic catarrh of the entire respiratory tract. Temporary asthma occurs during acute

congestive exacerbations, due to atmospheric changes and to derangement of the circulatory and digestive apparatus.

BOSWORTH asserts that a diseased condition of the nasal mucous membrane tends to produce disease of the bronchial mucous membrane. Asthma is due to a vasomotor paresis of the blood-vessels of the mucous membrane of the bronchial tubes. Polypoid degeneration, œdematous hypertrophy of the nasal mucous membrane, and nasal polypi indicate ethmoiditis. If we remove these conditions, we do not cure the asthma, which can only be remedied by radical treatment of the ethmoid, viz., to relieve the intracellular pressure by breaking down the trabecular walls by means of the burr. M. TOEPLITZ.

f.—NASO-PHARYNX.

348. LUZZATO. On the histology of the hypertrophic pharyngeal tonsil. *Arch. ital. di Otol.*, etc., vol. viii., p. 394.

349. LEWIN, L. Tuberculosis of the pharyngeal tonsil. *Arch. f. Laryng.*, vol. ix.

350. DE SIMONI. Adenoid vegetations according to the new views of Hertogue. *Bolletino delle mal. del'orecchio*, 1899, p. 491.

351. MICHALKIN, P. Treatment of a fibrous naso-pharyngeal polyp with electrolysis. *Medicinskoji obosrnj.*, No. 5, 1899.

352. INGALS, E. FLETCHER. Fibrous tumor of the naso-pharynx; sequel. *N. Y. Med. Jour.*, Dec. 16, 1899.

348. LUZZATO examined the peculiarities of the epithelium and observed emigration of leucocytes in well-preserved ciliated epithelium and in squamous epithelium, as opposed to Stöhr's view. The author could not confirm McBride and Turner regarding the flattening of the epithelium. A simple hypertrophy of the adenoid tissue was found in all (fifty) cases. There were no sclerotic areas, but numerous hemorrhages and cysts. In two tubercular changes were present; one of these caused tubercle in the guinea-pig. GRADENIGO.

349. LEWIN places the following questions: 1. With due regard to all the circumstances which govern the relationship of a process to tuberculosis, how often does tuberculosis hide itself under hyperplasia of the pharyngeal tonsil? 2. By histological examination of pharyngeal tonsils, removed post-mortem from phthisical subjects, how often are they tubercular? These are the conclusions: 1. In our experience tuberculous foci are present in

about 5 per cent. of cases of hyperplastic pharyngeal tonsils. 2. The tuberculosis is in the so-called tumor form of mucous membrane tubercle; it is characterized by the absence of all externally recognizable marks, the so-called latent tuberculosis of the tonsils. 3. This latent tuberculosis may be the first and only localization of tuberculosis in the patient. 4. It is usually associated with tuberculosis elsewhere, especially of the lungs, which may not be manifest at the time of operation. 5. It is a comparatively frequent condition in pulmonary tuberculosis. 6. It may attack normally large as well as hyperplastic tonsils. 7. It is a relatively unimportant factor in the etiology of pharyngeal tonsillar hypertrophy. 8. It can be definitely overcome by elimination of the tonsil even in simultaneous pulmonary affection.

This very careful paper was written with the guidance of Professor Brieger. ZARNIKO.

350. DE SIMONI endeavors with theoretic reasonings to connect adenoidism and thyroidism. He regards adenoidism and myxœdema as belonging to the same disease, from analogy of clinical symptoms of those possessing adenoids and of the weak-minded myxomatous, the presence of adenoids in the latter, and the great frequency of adenoid vegetations where cretinism is endemic. GRADENIGO.

351. A farmer, thirty years of age, had his nose completely filled with grayish-red, soft, bleeding polypi. The buccal and pharyngeal cavities were occupied by a hard, fleshy mass starting from the base of the sphenoid. The tumor on examination proved to be a soft angio-fibroma. The growth was removed by electrolysis in a course of treatment lasting seventy-three days. No relapse after eight months. SACHER.

352. A man, now aged twenty-eight, had a fibrous tumor of the naso-pharynx as a boy of thirteen years. INGALS had then removed the tumor except some part attached to the vertical plate of the palate bone. It began afresh to crowd out beneath the zygomatic arch. An attempt at radical removal through an outer incision in the cheek from mouth to ear had to be abandoned owing to profuse hemorrhage. The growth then continued to grow for about a year, completely closing the right nasal cavity and destroying the sight of the right eye. The tumor then remained stationary for many months, but the patient began in a couple of months to breathe a little through the nose. From now on he steadily improved, until after several years the nasal cavity ap-

peared perfectly free and the right cheek had grown smaller. The right eye appears normal, but is blind. The fibrous growth has disappeared. The right nasal cavity is an inch wide, the septum is pushed aside, and the turbinates are destroyed. There is a large opening in the sphenoid cells. M. TOEPLITZ.

SOFT PALATE, PHARYNX, AND BUCCAL CAVITY.

352 *a*. COUVELAIRE and CROUZON. Movements of the soft palate. Transactions of the Biological Society. *Le progrès médical*, Dec. 2, 1899.

353. MASINI, G. Have the tonsils an internal secretion? *Ann. des mal. de l'or., du lar.*, No. 7, 1899.

354. MAMLOK. A case of primary malignant lymphoma of the tonsil. *Arch. f. Laryng.*, vol. ix.

355. LJANZ. The treatment of mercurial stomatitis. *Medicinskoje Obosrenje*, Jan., 1899.

356. GOLDSCHMIDT. The smooth atrophy of the root of the tongue in tertiary syphilis. *Berl. klin. Wochenschr.*, No. 43, 1899.

357. L. A. The treatment of angina and diphtheria in Cælius Aurelianus. *Münch. med. Wochenschr.*, No. 47, 1899.

358. SIEGERT. On an epidemic of lacunar angina and its period of incubation. *Münch. med. Wochenschr.*, No. 47, 1899.

359. MAYER, EMIL. The tonsils as portals of infection. *Jour. Am. Med. Assoc.*, Dec. 28, 1899.

360. GOODALE, J. L. Acute suppurative processes in the faucial tonsils. *N. Y. Med. Jour.*, Oct. 7, 1899.

361. LELAND, GEO. A. Tonsillar and circumtonsillar abscess. *N. Y. Med. Jour.*, Oct. 7, 1899.

362. HUBBARD, THOMAS. Peritonsillar abscess associated with diphtheria. *N. Y. Med. Jour.*, Oct. 14, 1899.

363. WARD, M. R. Septic thrombo-phlebitis as a complication of peritonsillar abscess. *N. Y. Med. Jour.*, Oct. 14, 1899.

364. WATSON, ARTHUR W. Accessory thyroid gland at the base of the tongue. *N. Y. Med. Jour.*, Oct. 21, 1899.

365. INGALS, E. FLETCHER. Fibro-lipomatous tumor of the epiglottis and pharynx. *N. Y. Med. Jour.*, Dec. 9, 1899.

366. MCREYNOLDS, JOHN. Chronic recurring membranous pharyngitis. *Jour. Am. Med. Assoc.*, Dec. 2, 1899.

367. GAGE, GEO. C. Some of the dangers of acute pharyngeal abscess obviated by the use of a new trocar. *N. Y. Med. Jour.*, Dec. 16, 1899.

368. HOPKINS, F. E. Recurrence of the tonsil after excision. *N. Y. Med. Jour.*, Dec. 2, 1899.

369. MUSSON, EMMA E. Infective granulomata of the pharynx; glanders. *Jour. Am. Med. Assoc.*, Nov. 25, 1899.

352 *a*. The movements of the soft palate were observed in a man with a large defect in the orbital and nasal regions following an operation for carcinoma. 1. During inspiration with closed mouth the velum is slightly raised. 2. The palate also moves conjointly with the pharyngeal wall, whereby the naso-pharynx is shut off. This consists of (*a*) a raising of the soft palate to not quite the horizontal, or beyond the horizontal (incomplete or complete closure); (*b*) an advancing of the posterior pharyngeal wall which approaches the soft palate; the posterior median line and the upper wall of the pharynx remain immovable; (*c*) a protrusion of the salpingo-pharyngeal plica, forming upper and posterior supporting columns for the velum. The closure of the naso-pharynx is complete during swallowing, sucking, expiratory pressure in blowing and whistling; an incomplete closure takes place during coughing. During phonation, the degree of closure varies: (*a*) in pronouncing vowels, the closure increases from *a* to *e*, from *e* to *o* and to *a*, and from *u* to *i*; (*b*) in pronouncing the consonants, the closure varies according to the accompanying vowels; (*c*) for the consonants *m* and *n* the closure is very incomplete.

SCHWENDT.

353. The tonsils of dogs and calves were removed and extracts made with water or glycerine which was injected into the auricular vein of the rabbit. The exposed heart and the femoral vein then showed for some time a distinct slowing and strengthening of the heart activity. This action did not take place when the tonsils had been chronically or congenitally hypertrophied. MASINI regards the tonsils as internal secreting organs.

ZIMMERMANN.

354. A very careful analysis of a typical case of this rare condition. ZARNIKO.

355. LJANZ discusses the prevention of mercurial stomatitis and speaks of a number of tooth pastes and soaps. The best remedy for stomatitis is hydrogen peroxide (8–10 per cent); it is non-toxic, non-irritating, and very germicidal. The author

prescribes a 2 per cent. gargle. In large and many ulcers he employs iodoform in powder or ethereal solution. SACHER.

356. After an examination of two hundred cases of syphilis, GOLDSCHMIDT concludes that the smooth atrophy of the base of the tongue is not clinically a pathognomonic sign of tertiary syphilis, as it may be present in other conditions, either combined with a poor development of the tongue in general or when the rest of the tongue is well developed. MÜLLER.

357. An interesting historical paper by an anonymous writer. AURELIANUS possessed a long list of therapeutic measures, including intubation, of which he personally was not in favor. SCHEIBE.

358. The period of incubation is four days. The patient should be isolated, and the brothers and sisters should not be allowed to attend school until the fifth day has passed without infection. SCHEIBE.

359. After an elaborate review of the published cases, in which an angina was followed by articular rheumatism, severe general infections, metastatic abscesses, angina pectoris, bronchopneumonia, and other affections, MAYER narrates a case of his own observation in a young man æt. nineteen, who twenty-four days after an attack of acute follicular tonsillitis was seized with syncope and vomiting. After the endocarditic murmur had become fainter, symptoms of hemichorea of the right side, and finally also of the laryngeal muscles, developed, from which the patient completely recovered. M. TOEPLITZ.

360. Eight cases of acute amygdalitis with intrafollicular foci of suppuration showed: 1, the streptococcus more abundant than the staphylococcus, where the foci were numerous; 2, the foci in two cases *with*, in six cases *without* circumtonsillar inflammation; 3, the foci clinically to represent a severe infection; 4, no clinical signs by which the abscesses could be diagnosticated; 5, histologically: *a*, the foci to vary in size, number, and location; *b*, the fibrinous exudate in the crypts quite marked; *c*, in the cases with peritonsillar abscess, the connective-tissue spaces crowded with polynuclear neutrophiles. The conclusion may hypothetically be arrived at, that the pyogenic infection of the follicles is secondary to a previous infection of the crypts by the staphylococcus pyogenes. M. TOEPLITZ.

361. LELAND used for opening tonsillar and circumtonsillar abscesses the sickle knife, cutting lengthwise through the tonsil,

and introduces his sterilized index finger into the incision, thereby breaking up the diseased tissue in and around the tonsil. The abscess is thus found much quicker than by other methods, and the duration of the affection is much shortened, as is well illustrated by the reported cases. In some instances the deep-seated abscess had to be opened on the following day with the probe-pointed knife. M. TOEPLITZ.

362. Case 1 : a farmer, æt. thirty, had, after an acute amygdalitis, his right tonsil incised and pus evacuated. The next day both tonsils and pharynx were found to be covered with false membranes. Thirty-five hundred units of antitoxine did not prevent the membranes from invading the naso-pharynx, nares, and larynx. On the sixth day laryngeal stridor and extreme dyspnœa, purulent discharge from the throat, and ichorous flow from the nares ensued, associated with extreme swelling of the anterior cervical region suggestive of phlegmon. Tracheotomy was performed. The patient died after eighteen hours. The wife and two children also had diphtheria, but recovered. Case 2: the eldest son of a large family had sore throat, two young children mild amygdalitis, a younger daughter typical diphtheria ; another had quinsy. About four days later the one was moribund from diphtheritic toxæmia and the other had a large peritonsillar abscess with pseudo-membrane. The abscess was incised and much pus evacuated. M. TOEPLITZ.

363. WARD adds to three cases collected from literature two of his own observation. Case 1 : a woman, æt. thirty, felt at first pain in the left tonsil, and after three days presented a swelling of the right tonsil and marked tumefaction of the right side of the neck with chilly sensation. The tumefaction extended from the angle of the jaw down to the clavicle. Then pain in the right side of the chest, cough, expectoration tinged with blood, diarrhœa, vomiting, enlargement of spleen, and severe chills appeared. Incisions of the tumefaction and tonsils evacuated pus. Death ensued on the ninth day. The autopsy revealed thrombosis and thrombo-phlebitis of the internal jugular and the veins leading upward to the tonsillar plexus, a metastatic abscess in the middle lobe of the right lung, other foci in the apex and base, and great enlargement of the spleen. Case 2: a man, æt. forty-two, had, after opening of a left peritonsillar abscess, increased swelling of the left tonsillar region and the tissues of the neck resembling a cellulitis. Death followed soon. The autopsy showed a thrombo-

sis and thrombo-phlebitis of the internal jugular and multiple small abscesses of the kidneys. M. TOEPLITZ.

364. WATSON reports two cases of accessory thyroid glands at the base of the tongue. The first occurred in a woman, aged fifty, and occupied the lingual base from the epiglottis to the papillæ circumvallatæ, being an inch and a half long, an inch wide, and an inch thick; the second case was seen in a colored girl, æt. sixteen, who had felt the lumps for five years in her throat. It looked like the first case except for its ulceration. The diagnosis was made in both cases by the microscope.

M. TOEPLITZ.

365. INGALS'S patient, æt. twenty-eight, had difficulty in speaking, swallowing, and breathing, particularly in a recumbent posture. A smooth tumor filled the laryngo-pharynx, leaving only a small chink about a quarter of an inch wide at the left side. Stout steel wire, passed through a uterine ecraseur, succeeded in cutting it off in four pieces of $1\frac{1}{2}$: 1, $\frac{1}{2}$: $\frac{1}{2}$, $1\frac{1}{4}$: $\frac{1}{2}$ and $\frac{1}{2}$ inches respectively. The tumor had been attached to the upper portion of the right side of the epiglottis, to the right pharyngo-epiglottic fold, to a part of the base of the tongue, and to the right side of the pharynx. The first removed mass was a typical fibroma, another a fibro-lipoma, and the last large mass a lipoma. The right side of the epiglottis became adherent to the pharynx and to the base of the tongue without preventing deglutition.

M. TOEPLITZ.

366. The patient, a female, æt. nineteen, presented a membrane remaining one or two days when it spontaneously disappears, leaving the throat in apparently healthy condition, always covering the soft palate, sometimes also the centre pharynx, being pearly-white, with pin-hole perforations, recurring two or three times a week when not treated. It contained no diphtheria bacilli and no fungi. M. TOEPLITZ.

367. The point of the trocar is cone-shaped and a guard ferrule is placed half an inch from the point. The curve of the trocar adapts itself to the shape of the tongue. A Y-shaped tube is connected with the trocar through one limb, the other ending in a rubber bulb, while to the stem a glass bulb is attached, which also ends in a rubber tube closed with a clamp. If the flow of the pus is too thick, the clamp is closed and the rubber bulb when squeezed produces suction. If the flow is thin, the trocar can be used without the tubing. M. TOEPLITZ.

368. HOPKINS adds to one case of his own observation occurring in a girl æt. thirteen, in whom one tonsil had recurred four months after excision, the views of many authors widely differing as to the cause and frequency of recurrence, the smallest number being observed by laryngologists. M. TOEPLITZ.

369. A woman, æt. fifty-six, presented rapid enlargement of the tonsils; she lost flesh, but had extreme fulness of the neck, beginning at each side of the angle of the jaw, giving it a pouched appearance. Apart from the large tonsillar masses, a soft growth of the size of a black walnut filled the left half of the naso-pharynx, and lingual masses were seen in the glosso-epiglottic space. Removed portion of the left soft and friable tonsil was supposed to be a sarcoma. After radical removal of the masses improvement took place. In April, 1895, a year and a half after the operation, the lingual masses had increased, the faucial ones had also returned, and the vault had filled up again, also on the right side. In December, 1895, the diagnosis of glanders was made with the microscope. On March 17, 1896, the fauces and naso-pharynx were thoroughly cleared from the masses, whereupon the patient improved. Inoculations of six guinea-pigs produced orchitis and intestinal lesions covered with bacilli mallei. On March 27, 1896, intestinal disorders took place. The pharynx appeared well in June, but the patient died in September. No autopsy was held. M. TOEPLITZ.

BOOK REVIEWS.

VII. **Leçons sur les suppurations de l' oreille moyenne et des cavités accessoires des fosses nasales et leurs complications intra crâniennes.** By Dr. HENRY LUC, ancien interne des hôpitaux de Paris. Octavo-volume of 500 pages, with 28 figures in the text. Paris: J.-B. Baillière et fils, 1900.

The author publishes, in 26 leçons, the lectures he delivered at his clinique in Paris.

The first lecture gives a general view of the topography of the accessory cavities, their connections with the naso-pharynx, and their contiguity to the cranial cavity to which their suppurations frequently extend. He mentions the unique case of Westermayer, where even an empyema of the maxillary antrum, the latter alone being at some distance from the skull, after perforation of the upper posterior wall entered the skull through the upper part of the pterygo-maxillary fossa. He speaks of the transmission of the infection from one sinus to the other, of the lining membranes, the pyogenic microbes, and the diagnosis of the empyemas where the old objective signs (swelling, redness of the integument, pressure sensibility, escape of pus) had been essentially supplemented by the electric illumination through mouth, nose, and upper-inner corner of the orbit.

The *next two* lectures are devoted to *acute middle-ear suppuration*, of which the author gives an excellent description. We mention some points. He says: "I cannot well imagine that an acute suppurative otitis exists without a certain degree of concomitant antritis, but we should not speak of mastoiditis before the suppuration has spread into the mastoid *cells*" (p. 16). He emphasizes the grave signs otitis produces in small children, which fact, "perhaps, is explained by the more intimate circulatory connection between the ear and brain in the child" (p. 20). As to the terminations, he distinguishes six kinds: recovery; re-

covery with diminution of hearing; recovery with persistent perforation of the drum membrane; with mastoid complication; with intracranial infection; transition into the chronic state (p. 25).

The variations of this, the typical clinical picture, may be designated by prominent symptoms, and their etiology, as the *grippe form*, by its tendency to mastoid and intracranial complications; the acute *necrosing* form in the infectious diseases, scarlet fever, diphtheria, typhoid fever, measles; further, the peculiar course when *erysipelas* develops in an ear with ordinary otitis purulenta, during the regular course of which at once are noticed long and marked chills, temperature 105° F., later falling to the normal, these attacks repeating themselves for the next days, until the characteristic elevated border of the erysipelas ambulans shows that pyæmia is not the cause of these rapid changes. *Tuberculosis* (lack of pain), *syphilis* (inordinate degree of deafness by labyrinth complications), and *diabetes* (tendency to extensive destruction of bone in mastoid and surroundings) are discussed.

The treatment does not contain anything new. Early large paracentesis, removal of the pus by inflation (catheter or Politzer), drainage by the introduction of thin, round, long wicks of absorbent cotton or gauze, touching or even a little entering the perforation in the drum-head (Loewe). The wound should be dressed or cleansed at least once daily, the ear inflated, the meatus mopped out with absorbent cotton, then a few drops of carbolic acid 1 part, to glycerine 15 parts, instilled, and a drainage wick of gauze introduced again. The glycerine-carbolic-acid wash acting as an antiseptic and analgesic, favors the escape of the secretion by mixing with and diluting the pus. When the period of pain is over, this treatment is replaced by peroxide of hydrogen and insufflation of boric acid powder. The cleansing with a syringe is to be substituted for the above dressing, if the patient cannot be dressed by the physician every day. He recommends caution in its use. We would say that the chief remedy in a case of acute otitis media is *rest in bed.* This disease is important and requires care and nursing. Forcible inflation of the ear before or after the paracentesis should be omitted, just as injections, for we have seen aggravation of the disease follow their use immediately. The inflations are proper when the active inflammation is passed. We have no experience with the carbolic-acid glycerine drops; we depend chiefly on paracentesis, dry

treatment, rest in bed, and patience until the full recovery is obtained, for relapses and dangerous complications are rife.

The next subject which the author takes up and describes in full detail is *acute* and *chronic mastoiditis* (55 pages). He emphasizes the variations of the structure of the mastoid as determining to a great degree the clinical picture. The etiology, symptomatology, and treatment are well presented, particularly the opening of the mastoid. He devotes a full lecture to the Bezold mastoiditis, which the peculiar features and the gravity of this variety fully deserve.

Lectures VII.–X. treat of chronic suppurative middle-ear inflammation (70 pages). The first lecture consists in general remarks on the disease, its causes, otoscopic condition (perforations of membr. tymp., small or large, the importance of their location, the aspect of the "fundus of the ear," the mucous membrane, congestive swelling, thickening, fungosities, granulations, and polypi, and their histology), epidermization, cicatrices, changes in the ossicles, etc. In the symptomatology he describes also the manner of examining the ear, its cleansing (Hartmann's tympanum syringe), and the significance of the substances which are removed, for instance those from the attic by the variable prognosis of facial paralysis and the acuteness of hearing, and the value of the exploration with the straight and bent probes. To judge how much importance as to prognosis and indications is to be laid on the different conditions found by a thorough examination, he describes them in five progressive types.

Lecture X., the suppuration of "Shrapnell's cavity." The author describes the attic, adopting the views of Schmiegelow as published in the *Zeitschrift für Ohrhke.*, 1891, and the English edition, these ARCHIVES, XX., p. 228–256. The various important conditions, caries, necrosis, granulations, polypi, and cholesteatoma, found in this small and intricate cavity are well set forth, and their treatment, up to ossiculectomy and removal of all carious and necrosed portions of the osseous walls, is dwelt upon.

The next three lectures are devoted to the consideration of *chronic mastoiditis*, 36 pages. The cases known as latent mastoiditis (no fistula, etc.) require careful examination of the tympanic cavity and its recesses, and judicious appreciation of the subjective and objective symptoms in the course of the affection, facial paralysis, etc. Deep, intense, constant pain, varying in intensity, and mostly pressure sensibility in a particular point,

mostly at the base, are the only signs preliminary to an intracranial complication.

The so-called radical operation, the opening of all the cavities of the ear, is described ; first Stacke's, then Zaufal's method. The descriptions are very clear, and the propositions well considered.

Cholesteatoma occupies a full lecture. The diversity of opinion on this remarkable formation is set forth at great length. The subject is practically very important.

Lectures XIV.–XIX. treat of the empyemas of the accessory sinuses (141 pages).

The maxillary sinus receives 47 pages ; the descriptions are very elaborate. He says an exact diagnosis begins with the cultivation of rhinology in modern times. The sign of Heryng (of Warsaw), shown first at the congress of Paris in 1889, namely, the translumination,[1] has assisted materially in the diagnosis of all the sinuses. It has been extended by Vohsen, Davidson, and others. Luc describes in full detail, and with a certain degree of enthusiasm, his way of curing chronic maxillary empyema ; he calls it La méthode opératoire Caldwell-Luc, because Dr. Geo. W. Caldwell, of New York, has published essentially the same operation before him (*New York Med. Jour.*, Nov. 4, 1893), of which the author heard only a year ago. The technique is as follows: 1. Incision of the mucous membrane of the mouth in the canine fossa in a horizontal line. 2. Chiselling through the bone horizontally at the level of the molar teeth as far as the angle between the lower and nasal walls. 3. Cleaning out the sinus with bent spoons. 4. Formation of an artificial hiatus in the nasal wall. 5. Establishing drainage into the nose. 6. Suturing the wound in the mouth. He has done this operation many times, and his colleagues in Paris have adopted it. The results have been rapid and permanent, exceedingly satisfactory recoveries. We cannot enter into further details, but have received the best impression from reading the description of the method and the accounts of recovery given by the author.

The *frontal-sinus* empyema is discussed at length, its simultaneous existence with ethmoidal and maxillary empyemas is emphasized, and, in chronic cases, the operation by removal of the anterior osseous wall recommended. He considers critically the

[1] We leave the French word "translumination" (Durchleuchtung in German), which is, perhaps, as good or better than the customary word, "transillumination," of English writers.

different methods. In rebellious cases, he says, a German surgeon, G. Dünt,[1] has proposed the total resection of the anterior wall. The author might have mentioned the osteoplastic operation of Czerny (Heidelberg) and Golovin (Moscow), and the bold and very excellent method of Jansen (Berlin). Jansen detaches the skin and periosteum along the inner corner and upper margin of the eyelid, together with the uncut pulley of the tendon of the superior oblique muscle of the eye, removes the lower bony wall of the sinus and all diseased bone, not only in the walls of the frontal sinus, but of the adjacent ethmoidal cells. The reviewer has seen the most surprisingly good results of this operation done in New York, and has adopted it himself.

The reviewer, greatly interested in Dr. Luc's monograph, has given his pen more scope than is usual in book reviews. He has to be brief with the remainder of the work. The empyemas of the ethmoidal and sphenoidal sinuses are described with the same care and judgment as the preceding subjects, which shows that the author is less of an "*autodidacte*" than he alleges to be in the preface of his book. He shows that he is fairly familiar with the literature of his subject, in particular the German, less perhaps than he should be with the English, but he is fully at home among the host of important diseases which form the subject-matter of his lectures. This can particularly be said of the last part of the work : the intracranial complications of the suppurative diseases of the middle ear and the accessory cavities of the nose.

The subjects of the remaining seven lectures are as follows : Mechanism and propagation of intracranial infection. Extradural abscess. Sinus thrombosis. Pyæmia without sinus-thrombosis. Brain abscess. Leptomeningitis. Further, a supplementary lecture on the ophthalmoscopic diagnosis of the cerebral complications of the sinusites, by Dr. Valude, of Paris.

The presentation of this last part of the book is in keeping with the preceding. The style of the book is clear and easy. It will introduce the student thoroughly into this important and essentially modern branch of medicine and surgery, and delight the adept by walking pleasantly over a familiar field in which the author points out to him many view-points the beauty and significance of which he probably did not appreciate before. H. K.

[1] The reviewer does not know this name : perhaps it is a typographical error for Kuhnt, Professor of Ophthalmology in Koenigsberg, who published, about three years ago, an excellent monograph on *Frontal-Sinus Empyema*.

VIII. **A Treatise on Nasal Suppuration.** By Dr. L. GRÜNWALD (Munich). Translated from the second German edition by WILLIAM LAMB, M.D., etc., Birmingham. Published by William Wood & Co., New York. Pp. 335. Price, $3.

The importance of affections of the accessory sinuses has of late years been more and more appreciated. The knowledge of this chapter of rhinology received its foundation by more exact and careful anatomical and pathological investigations. The clinical aspect has been furthered especially by Grünwald. *Die Naseneiterungen* of this author was the first—and until recently the only—book giving a detailed description of these affections. Its excellence and deserved popularity are well known. Its inaccessibility to those not conversant with German has now been removed by the appearance of Dr. Lamb's translation.

The localized or focal suppurations of the nose and its accessory cavities are treated in a general and in a special part. In the former, the etiology, morbid anatomy, symptomatology, methods of examination, therapeutics, and prognosis are discussed in general. In the second part, the suppurations are taken up separately, and the special features in each variety are dwelt upon. The subject-matter is illustrated by case histories from the author's practice, and frequently by a critical review of the cases published by others bearing on the subject in question. The methods of treatment are described in an especially lucid and practical manner. The relation of syphilis to nasal suppuration and a very brief chapter on tuberculosis are added in an appendix. A complete bibliography up to the year 1896 (the date of the last German edition) concludes the volume.

The work of the translator has been extremely well done. Dr. Grünwald's very vigorous and interesting style seems not to have lost force in the translation. As far as the book itself is concerned, it is excellently gotten up and quite surpasses the German original. We are sure that in its enlarged field of activity this book will continue to instruct, and stimulate investigation, in this very interesting field of nasal surgery. A. K.

APPOINTMENTS.

NEW YORK POLYCLINIC: Drs. Francis J. Quinlan and R. C. Myles have been elected Professors of Laryngology and Rhinology at the New York Polyclinic.

SOCIETY MEETINGS.

The Western Ophthalmological and Oto-Laryngological Society elected, at their last meeting, April 5–7, 1900, at St. Louis, Mo.: Dr. M. A. Goldstein, of St. Louis, President; Dr. H. V. Wuerdemann, of Milwaukee, First Vice-President; Dr. C. R. Holmes, of Cincinnati, Second Vice-President; Dr. Fayette C. Ewing, of St. Louis, Third Vice-President; Dr. W. L. Ballenger, of 100 State Street, Chicago, Secretary. The place and time of the next meeting will be Cincinnati, O., April 11–12, 1901.

We are glad to publish the following notice in compliance with the request of the editors of the *Journal of Laryngology, Rhinology, and Otology:*

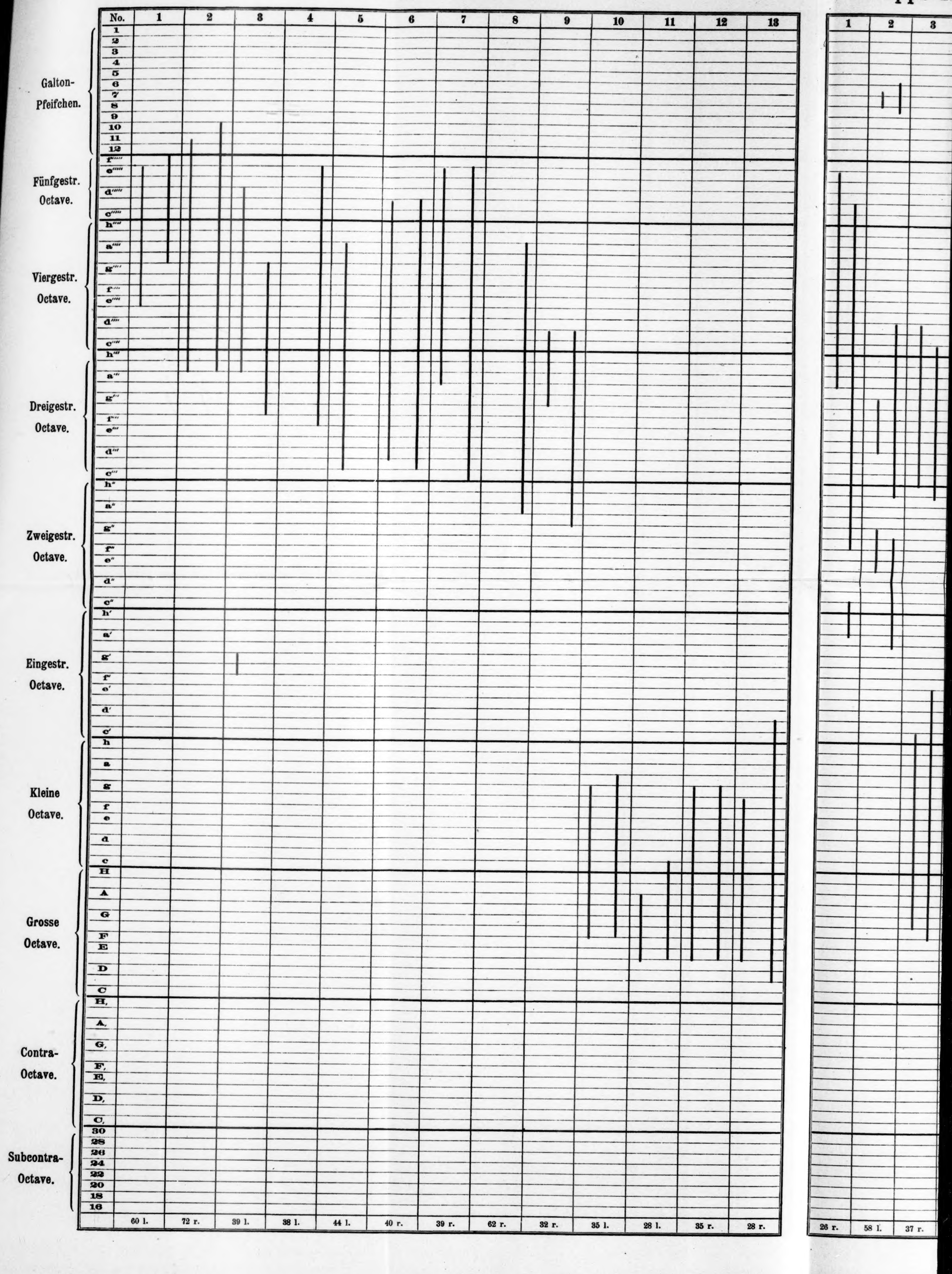
"An Appendix to the *International Directory of Laryngologists and Otologists*, compiled by Mr. Richard Lake, is in course of preparation. In it will be found corrections of names and addresses already given, an additional list of names and addresses received since publication, and an obituary list.

". . . Considerable additions have been obtained for the foreign list, which will materially add to its value and completeness. The decision of the editors of the *Journal of Laryngology, Rhinology, and Otology*, under whose auspices the Directory is published, to allow no name to be inserted in the British list for which sanction has not been given in writing, at once explains some omissions and criticisms. The editors, whilst desirous of making the Directory as complete as possible, consider it best to adhere to this course. It is therefore hoped that all engaged in the practice of Laryngology, Rhinology, and Otology will assist as far as possible in making this useful work complete," by sending in their names and addresses to the editor, *International Directory of Laryngologists and Otologists*, 129 Shaftesbury Avenue, W. C., London.

Die Tonstrecken der Taubstummen 1893 und 1896.

Gruppe I, Inseln.

Gruppe II



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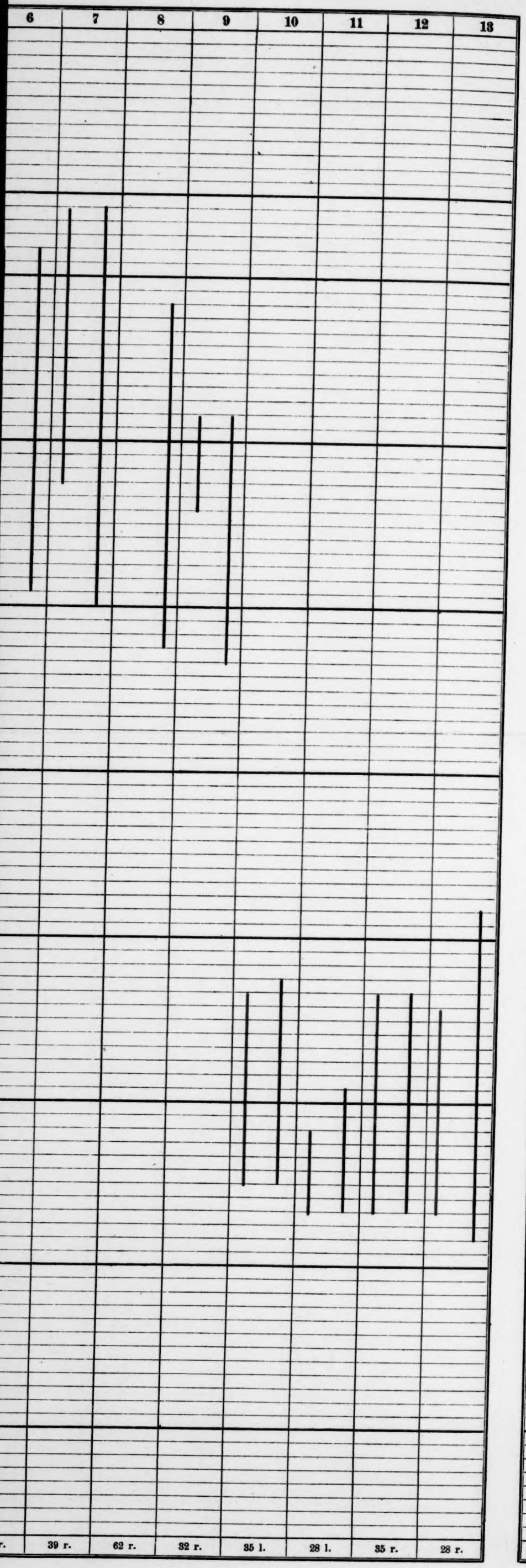

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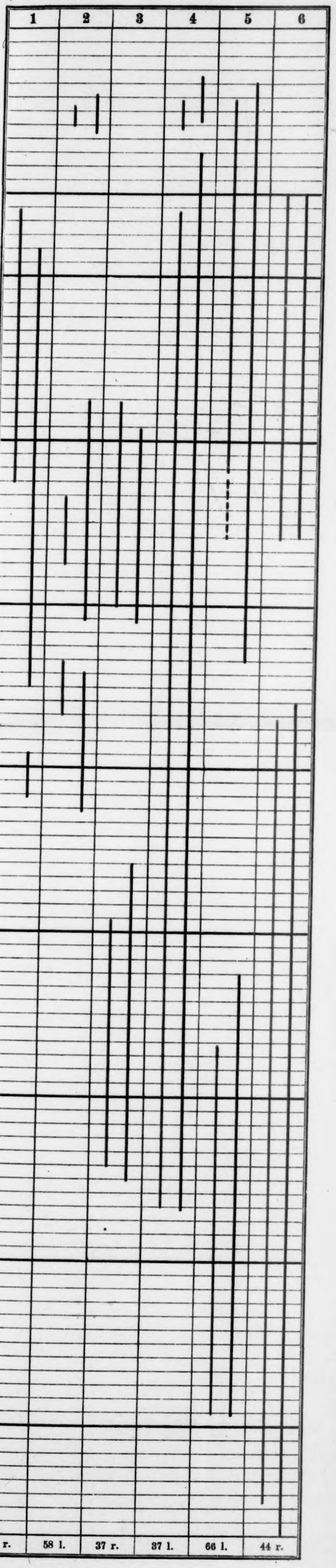

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Tafel 2.

Die Tonstrecken der Taubstummen 1893 und 1896.

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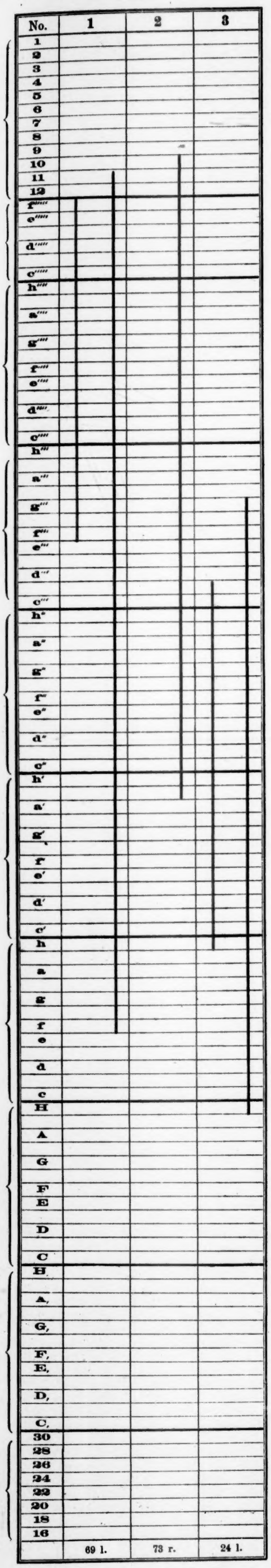

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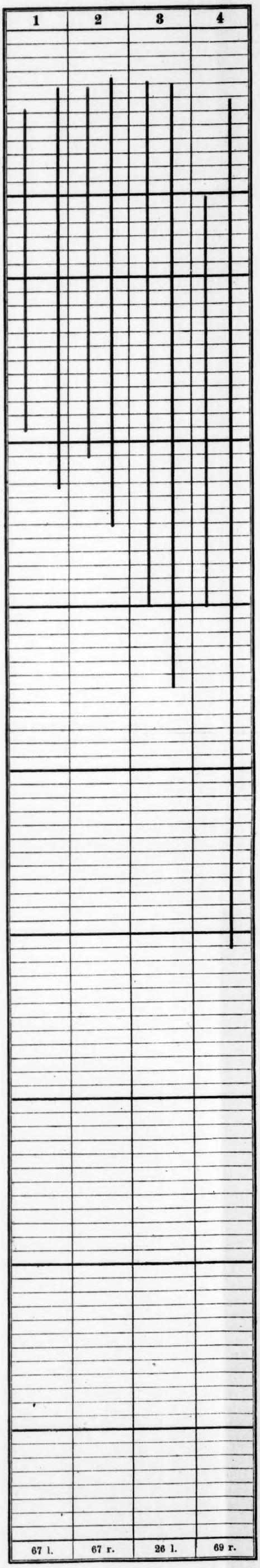

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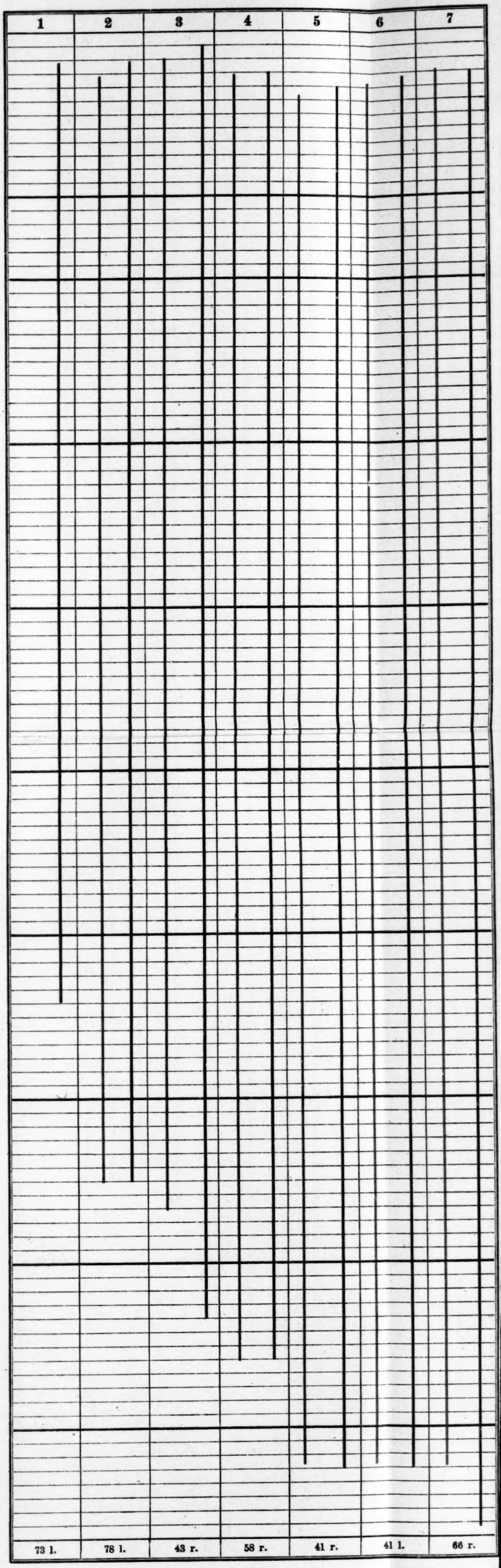

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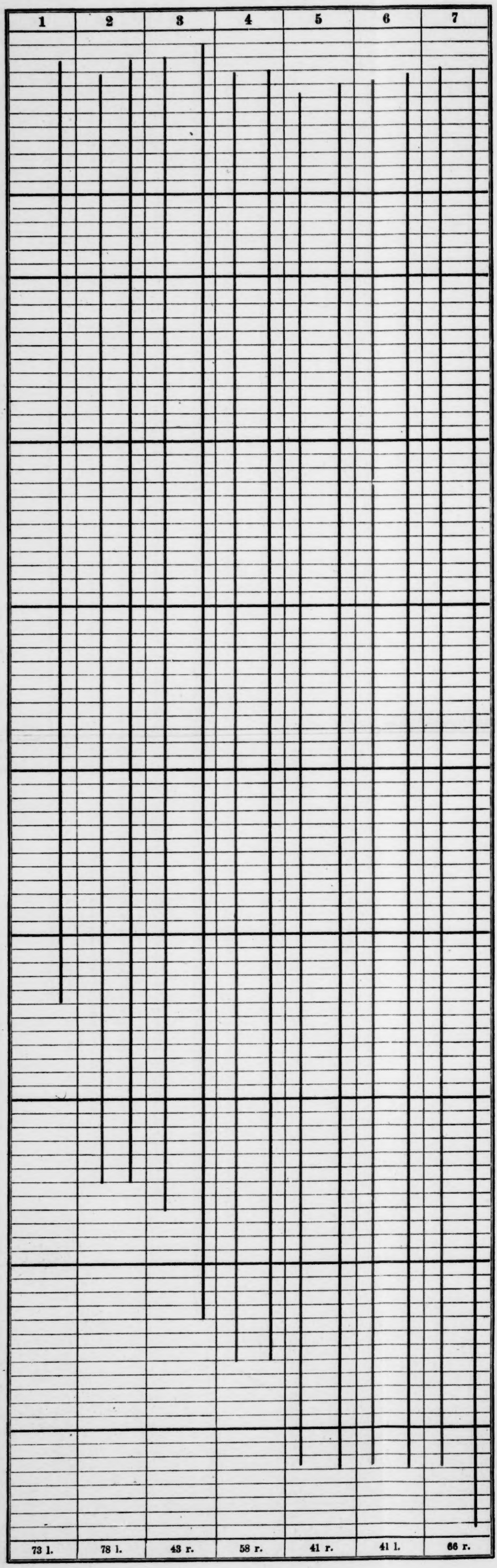

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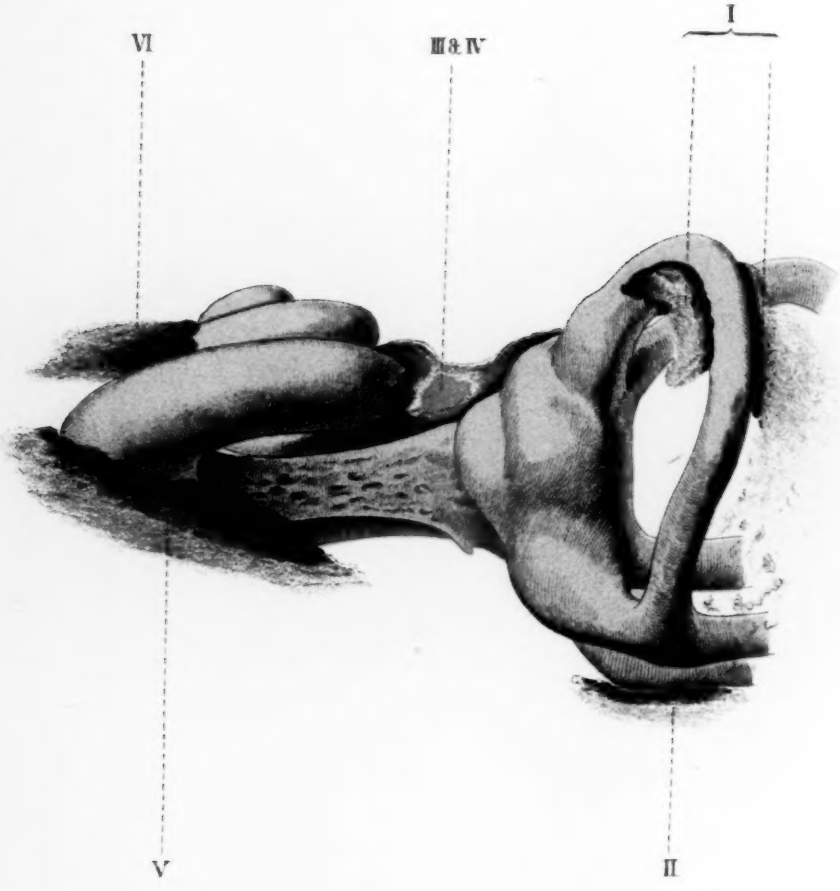

C. Krapf' lith.

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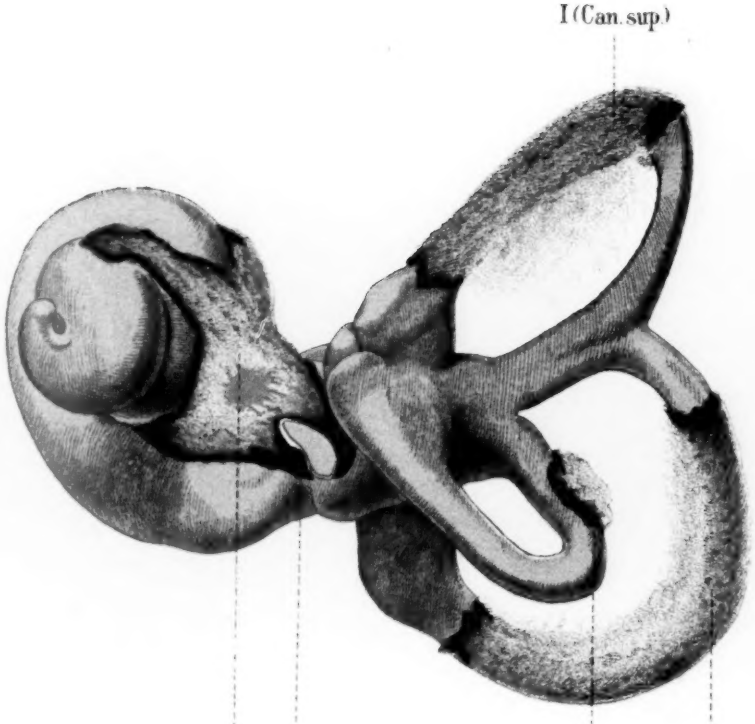

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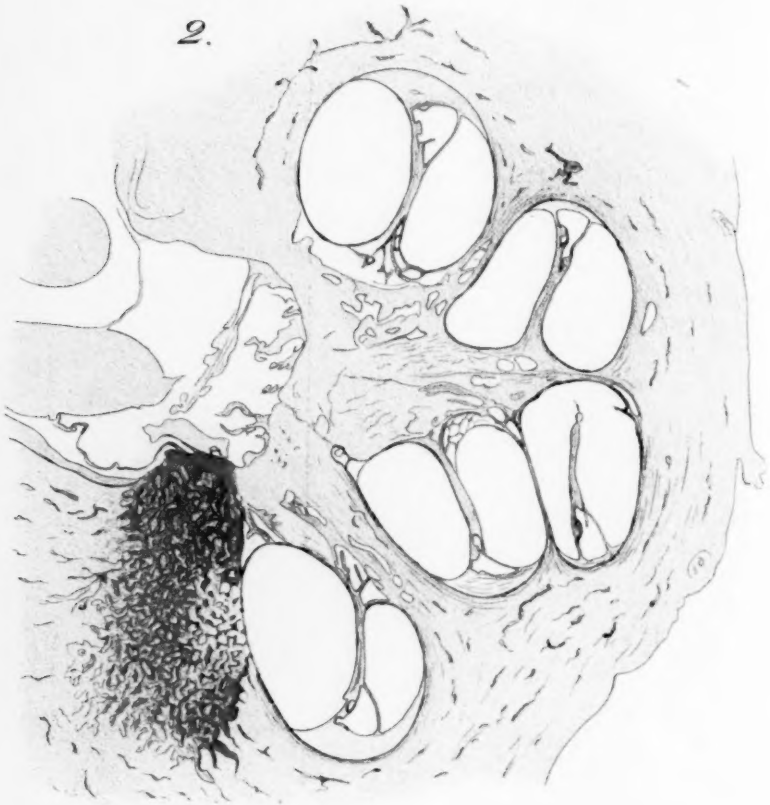

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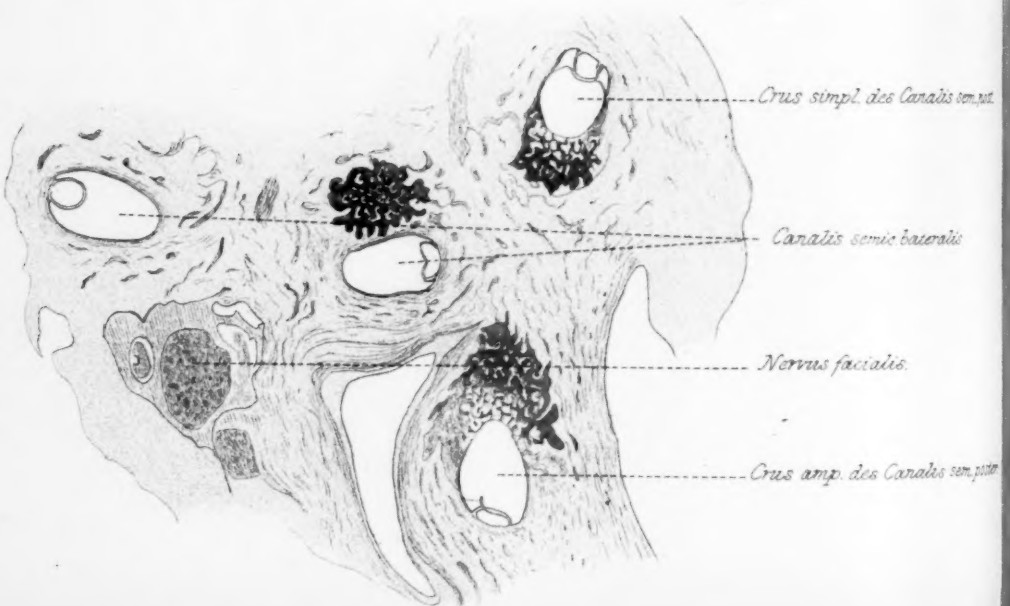

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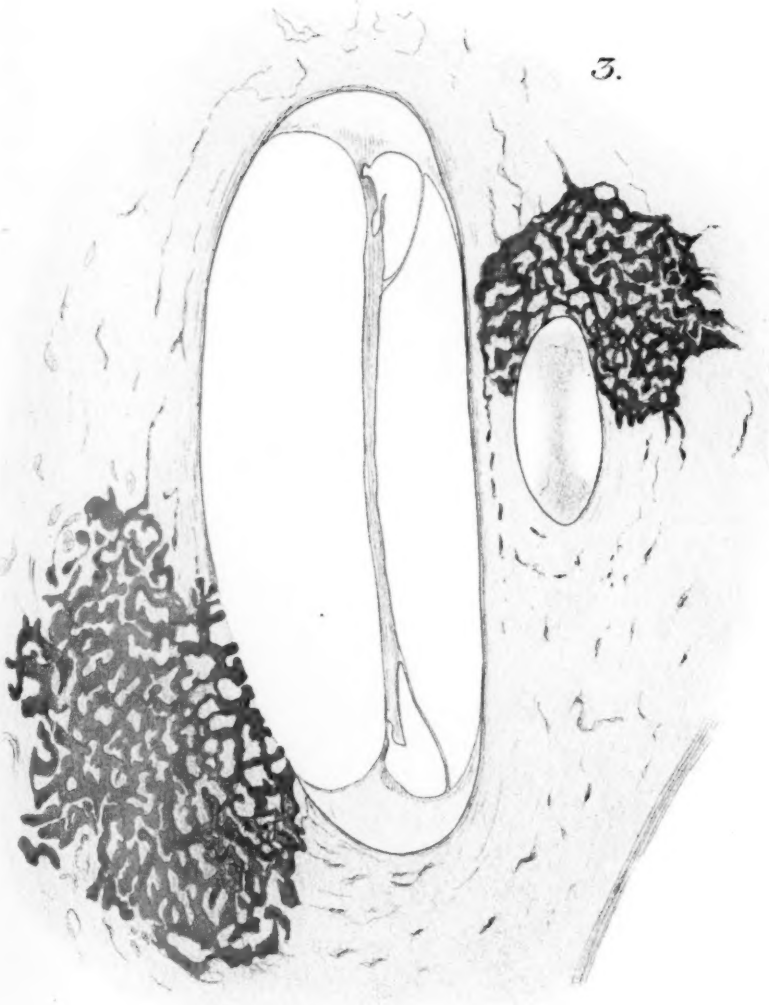

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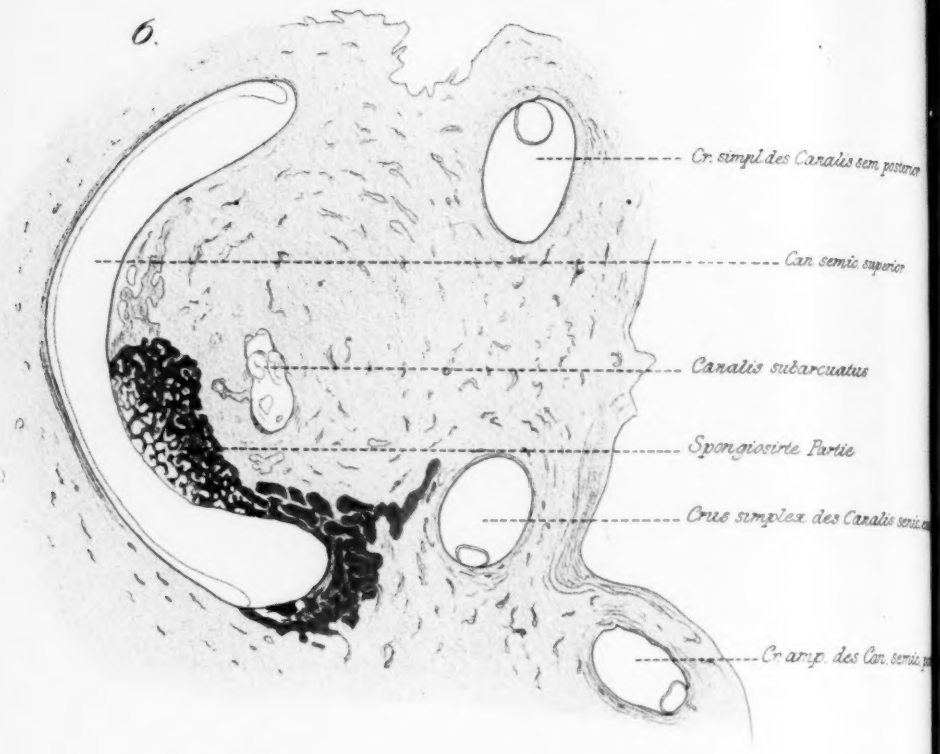

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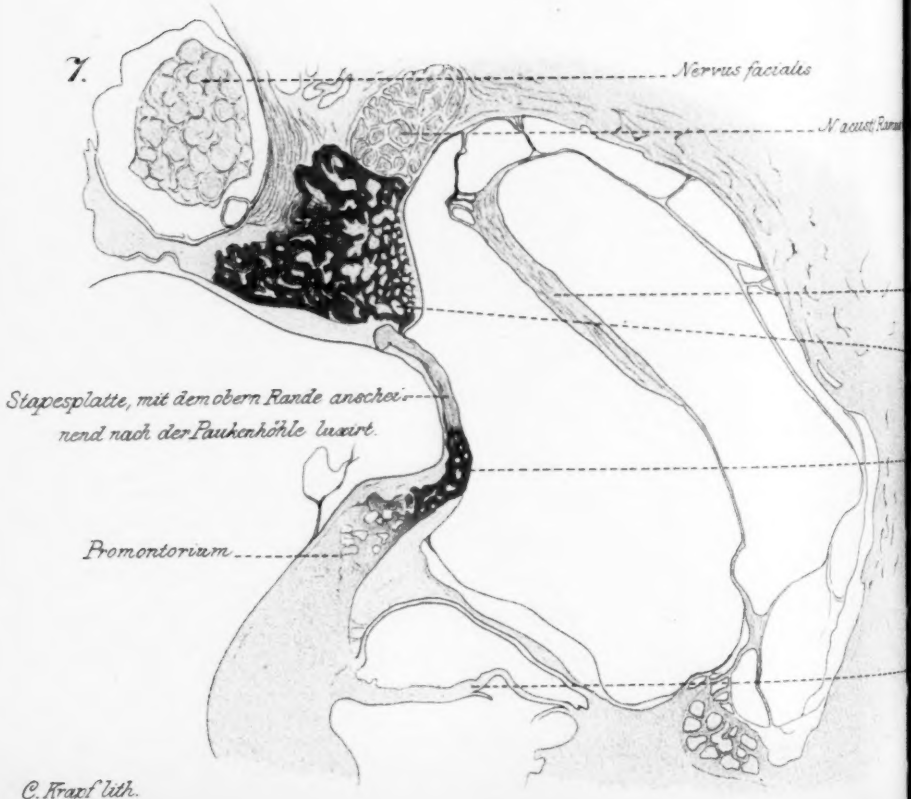

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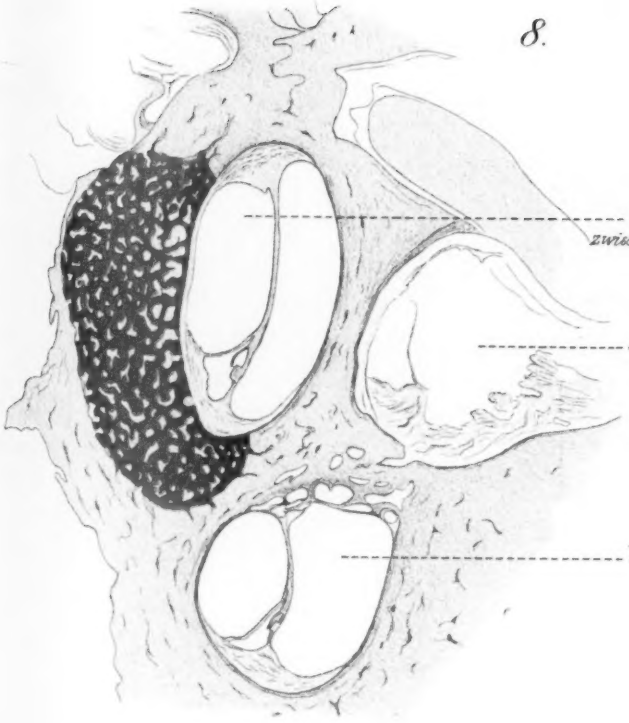

...ulo-ampullaris)

...Macula utriculi

...Oberer Umfang des ovalen Fensters
...pongiosirt u. wallartig nach dem Vestibulum zu verdickt.)

...Unterer Rand der Stapesplatte spongiosirt u. mit dem ebenfalls
...pongiösen untern Fensterrahmen knochern ankylosirt.

...Tympanum secundarium.

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